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ALL are in agreement on the need of building up the best veterinary service this country is able to maintain.

If opinions differ in significant details as to what constitutes a good veterinary service, all are in accord as to the importance of developing an efficient liaison between science and practice: between the fields of research and investigation and the farm.

As there are conflicting interests in the present complex constituting the veterinary service, would it not be of national benefit for the various branches comprising animal conservation and production to come to a common understanding?

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Vol. LXXXVI, N. S. Vol. 39 MAY, 1935 No. 5

RABIES LEGISLATION

Under date of March 26, 1935, the General Assembly of North Carolina ratified "an act (H. B. 174) to prevent rabies" in that state. This law consists of 25 sections, and a careful reading of it reveals that it is a rather unusual piece of legislation.

The act provides for the compulsory annual vaccination of all dogs in North Carolina against rabies, and also for the appointment of a "rabies inspector" in each township, these appointments to be made either by county health officers in those counties regularly employing such officials, or by the county commissioners in those counties where health officers are not employed. It is provided further that in the designation of "rabies inspectors," preference would always be given to licensed veterinarians living in the township. Under certain prescribed conditions, a veterinarian may act as rabies inspector for any township adjoining the one in which he lives.

The new law provides that the owner of each dog shall pay no more than 50 cents to the rabies inspector for the vaccination, the amount to be credited on the dog tax when a certificate of vaccination is presented to the sheriff or the tax collector of the county. The rabies inspector is authorized to "retain for his service the sum of 25 cents for each dog vaccinated." The balance is to be remitted to the State Department of Agriculture,

to be used for covering the cost of the vaccine and other expenses incidental to the enforcement of the law.

Two sections of the law provide for quarantines against rabies by the Department of Agriculture if same should be necessary in the event of an outbreak of the disease. Other sections provide for killing rabid animals or those known to have been exposed to rabid animals. Provisions are made also for quarantining animals suspected of having rabies.

The veterinarians of North Carolina are not particularly pleased with the new law. A special meeting of the North Carolina State Veterinary Medical Association was held at Raleigh on April 13, with half of the membership in attendance. It was decided by an overwhelming majority to have nothing to do with the bill. According to a communication received from Dr. J. H. Brown, secretary-treasurer of the Association, the following motion prevailed:

> That the veterinarians of North Carolina do not accept appointment as rabies inspectors; that $1.00 or more be charged for vaccinating dogs with rabies vaccine; and that rabies vaccine be bought only from those veterinary supply houses that sell exclusively to the veterinarian.

It would appear that this law is an open invitation to wholesale quackery in the state of North Carolina. It is estimated that there are in the neighborhood of 1,450 townships in the state. If each township is to have a rabies inspector, as provided in the law, it would be necessary to have about 1,450 rabies inspectors, less the number of cases in which a veterinarian could act as the rabies inspector for an adjoining township besides his own. There probably are not more than 100 graduate practitioners in North Carolina at the present time. The total number of veterinarians in the state, including those engaged in an official capacity, is not over 125. This means only one thing. If the provisions of the act are carried out, a large number of incompetent "rabies inspectors" will be appointed and North Carolina will have a new form of legalized quackery. The physical nature of rabies vaccine is such that more skill is required in its administration than is the case with almost any other single-dose biological product. We feel sorry for any dogs that are injected with rabies vaccine by any person other than a graduate veterinarian.

There is even some question of the propriety of requiring the annual vaccination of all dogs in a territory embracing over 52,000 square miles. There might be good reason for requiring the vaccination of dogs against rabies in such a large territory occasionally, or there might be some necessity for vaccinating

all the dogs in a smaller territory several years in succession. In each case, it is understood that proper recognition will be given to adequate quarantine measures.

Section 6 of the act provides that a vaccination tag shall be worn at all times by the dog to which it is issued, but the law does not say just how this requirement will be enforced. We have known dogs that had a way of losing their tags just about as fast as new ones could be put on, and there also have been numerous reports of the stealing of tags from vaccinated dogs and of their being attached to unvaccinated dogs by unscrupulous individuals. We anticipate that sheriffs and rabies inspectors in North Carolina will have their hands full when the time comes to check up each year, as provided in Section 8. It looks like another noble experiment.

A Section of Downtown Oklahoma City of 1935.

OKLAHOMA CITY EXTENDS WARM WELCOME

From a "water tank" stop on a prairie railroad on the morning of April 22, 1889, to a population of 10,000 by nightfall of that same day, Oklahoma City has forged ahead so rapidly that a visitor who knew nothing of its exciting history would find it hard to believe that this modern, bustling metropolis of 200,000 souls is only 46 years old.

Beginning as a city of visitors, Oklahoma City has been friendly to visitors ever since and, in recent years, has been increasingly recognized as one of the major convention cities of America. It is now making its plans to give a hearty welcome to members of the A. V. M. A. when they assemble for their annual meeting, August 27-30. It will have many things to show the visiting veterinarians: well-managed farms; blooded live stock; mammoth oil wells; beautiful parks; large industrial plants; comfortable homes; towering skyscrapers, and hotels that rank with the best.

The Oklahoma City Chamber of Commerce, through its Conventions Division, joins Oklahoma veterinarians in assuring members of the American Veterinary Medical Association a cordial welcome to the city that "daring built."

COMMITTEE ON LOCAL ARRANGEMENTS

Oklahoma veterinarians are now fully organized for the A. V. M. A. convention which will be held in Oklahoma City, August 27, 28, 29 and 30. The Committee on Local Arrangements, headed by Dr. C. C. Hisel, State Veterinarian of Oklahoma, has been holding monthly meetings, and it is probable that, as the time of the convention draws near, more frequent meetings will be found necessary. Dr. C. H. Fauks, of the local U. S. B. A. I. staff, and secretary of the Oklahoma Veterinary Medical Association, is in the responsible position of secretary of the Committee. Dr. Hisel and Dr. Fauks have been engaged in a friendly race to see who could secure the larger number of applications for membership in the A. V. M. A. To date the honors are about even. The complete personnel of the Committee follows:

Chairman: C. C. Hisel, Capitol Bldg., Oklahoma City.
Secretary: C. H. Fauks, 1719 S. W. 15th St., Oklahoma City.
Alumni: L. B. Barber, 334 Federal Bldg., Oklahoma City.
Army Relations: J. H. Wirtz, 1604 Euclid, Oklahoma City.
Banquet and Entertainment: J. T. Wilson, Pawnee.
Clinics: S. L. Stewart, 225 N. Pine St., Olathe, Kan.

CHAIRMEN OF SUBCOMMITTEES ON SPECIAL CLINICS

Cattle: W. H. Martin, El Reno.
Horses: G. A. Cunningham, Lawton.
Poultry: H. W. Orr, Oklahoma A. and M. College, Stillwater.
Sheep: R. W. Bowerman, 100 Live Stock Exchange Bldg., Oklahoma City.
Small Animals: H. W. Ayers, Box 848, Oklahoma City.
Swine: E. W. Meads, Claremore.

Exhibits: L. J. Allen, 1610 N. Ellison, Oklahoma City.
Finance: F. Y. S. Moore, McAlester.
Hotels: C. H. Fauks, 1719 S. W. 15th St., Oklahoma City.
Ladies' Entertainment: C. H. McElroy, Oklahoma A. and M. College, Stillwater.
Registration: W. L. Hiatt, Edmond.
Transportation and Parking: S. R. Espy, 123 N. W. 23rd, Oklahoma City.

It will be noted that Dr. S. L. Stewart, of Olathe, Kan., has been drafted by the Oklahoma veterinarians to direct the clinics. Dr. Stewart recently made the statement that the Oklahoma City clinic would be the biggest and best in the history of the A. V. M. A. The Oklahoma City Coliseum has been secured for holding the clinics.

EXECUTIVE BOARD ELECTIONS

As announced in the April issue of the JOURNAL, the polls for the primary elections in Executive Board Districts 5 and 7 were closed on April 27 and the ballots counted to determine the nominees in each district. Ballots were prepared and mailed on April 29 to all members in good standing in both districts. The names of the nominees, with brief statements relative to their A. V. M. A. activities, follow.

The ballots for the election proper in District 5 contained the names of only four nominees, as Dr. C. H. Stange previously had notified the secretary of the A. V. M. A. that he did not care to be a candidate even though he might secure sufficient votes to place him among the high five. On the other hand, the ballots mailed to members in District 7 contained the names of six nominees. This is because there was a three-cornered tie for fourth place, a contingency covered in Section 1 of Article 10 of the By-laws. The polls for the election will remain open until June 27.

The death of Dr. C. A. Cary, of Auburn, Ala., on April 23, created a vacancy in the Executive Board for District 4 (Alabama, Cuba, District of Columbia, Florida, Georgia, Kentucky, Maryland, Mississippi, North Carolina, South America, South Carolina, Tennessee, Virginia, West Indies and West Virginia). President MacKellar will appoint a member of the Board to serve District 4 temporarily and until such time as a special election can be held to select a successor to Dr. Cary. Ballots calling for nominations already have been mailed to all members in good standing in the district, as provided in Section 9 of Article V of the Constitution.

DISTRICT 5

KERNKAMP, H. C. H. — Saint Paul, Minn.

Associate Professor of Veterinary Medicine, University of Minnesota. Graduate of Ohio State University, 1914. Joined A. V. M. A., 1916. Chairman of Section on Education and Research, 1923-24. Resident secretary for Minnesota, 1924-27, 1928-31 and 1934-35.

PARKER, F. F. — Des Moines, Iowa

Practitioner. Graduate of Iowa State College, 1900. Joined A. V. M. A., 1924. Chairman of Section on Small Animals, 1930-31. Member of Executive Board, 1934-.

SEIDELL, H. A. — Des Moines, Iowa

State Veterinarian of Iowa. Graduate of Iowa State College, 1917. Joined A. V. M. A., 1919.

STANGE, C. H. — Ames, Iowa

Dean, Division of Veterinary Medicine, Iowa State College. Graduate, Iowa State College, 1907. Joined A. V. M. A., 1907. Member of Executive Committee, 1915-16. Member (1917-19, 1925-28) and chairman (1919-20, 1928-30) of Executive Board. President, 1923-24. Member of Committee on Policy, 1928-30. Member of Special Committee on Program for the American Association for the Advancement of Science meeting, 1929-30. Member of Committee on International Veterinary Congress, 1930-34. Member of Committee on Education, 1933-.

WEST, R. L. — Waseca, Minn.

Practitioner. Graduate of Kansas City Veterinary College, 1918. Joined A. V. M. A., 1919. Member of Executive Board, 1933-34.

DISTRICT 7

BRANDENBURG, T. O. — Bismarck, N. Dak.

State Veterinarian of North Dakota. Graduate, New York State Veterinary College at Cornell University, 1913. Joined A. V. M. A., 1915. Member of Special Committee on Tuberculosis, 1933-.

EHMER, E. A. — Seattle, Wash.

Practitioner. Graduate, State College of Washington, 1918. Joined A. V. M. A., 1919. Member of Committee on Local Arrangements, Portland meeting, 1924-25. Chairman of Section on Small Animals, 1924-25.

HAYS, C. H. — Pierre, S. Dak.

U. S. B. A. I. Inspector-in-Charge. Graduate, Ohio State University, 1908. Joined A. V. M. A., 1916. Member of Executive Board, 1930-.

LYTLE, W. H. — Salem, Ore.

State Veterinarian of Oregon. Graduate, Iowa State College, 1902. Joined A. V. M. A., 1910. Member of Committee on Army Service, 1918-19. Member of Committee on Veterinary Biological Products, 1924-25. Secretary (1927-28, 1929-31) and chairman (1931-33) of Section on Sanitary Science and Food Hygiene. Resident secretary for Oregon, 1928-29 and 1931-33. Member of Committee on Program, 1929-31.

SIMMS, B. T. Corvallis, Ore.

Professor of Veterinary Medicine, Oregon Agricultural College. Graduate, Alabama Polytechnic Institute, 1911. Joined A. V. M. A., 1912. Resident secretary for Oregon, 1916-24. Member (1919-23, 1924-25, 1926-28) and chairman (1923-24, 1925-26) of Committee on Intelligence and Education. First vice-president, 1927-28; fourth vice-president, 1930-31. Chairman of Committee on Resolutions, 1928-31. Member of Committee on International Veterinary Congress, 1931-34. Secretary of Section on Research, 1933-. Member of Committee on Program, 1933-.

WEGNER, E. E. Pullman, Wash.

Dean, College of Veterinary Medicine, State College of Washington. Graduate, State College of Washington, 1908. Joined A. V. M. A., 1913. Member of Committee on Local Arrangements, Portland meeting, 1924-25. Member of Committee on Resolutions, 1924-25 and 1929-30. First vice-president, 1925-26. Resident secretary for Washington, 1928-29.

APPLICATIONS FOR MEMBERSHIP

(See January, 1935, JOURNAL)

FIRST LISTING

BERENS, ROBERT 9247 Queens Blvd., Elmhurst, Long Island, N. Y.
V. M. D., University of Pennsylvania, 1931
Vouchers: Frank Bloom and Hyman W. Gardner.

BONACI, ALEXANDER J. 2420 Fourth Ave., Seattle, Wash.
B. V. Sc., Ontario Veterinary College, 1934
Vouchers: S. W. Clark and E. A. Ehmer.

BOYCE, LT. ROBERT A., JR. Quartermaster Remount Depot, Front Royal, Va.
V. M. D., University of Pennsylvania, 1931
Vouchers: Maj. William H. Dean and Capt. E. W. Young.

BURGETT, MARION V. 1122 W. 143rd St., East Chicago, Ind.
D. V. S., Kansas City Veterinary College, 1911
Vouchers: Glenn L. Ebright and E. L. Quitman.

CHRISTY, WILLIAM L. Tonkawa, Okla.
V. S., Indiana Veterinary College, 1901
Vouchers: C. C. Hisel and L. J. Allen.

COANE, SAMUEL 1841 Greenwood Ave., Trenton, N. J.
V. M. D., University of Pennsylvania, 1909
Vouchers: Arthur D. Goldhaft and Frank E. Lentz.

DOLAN, RICHARD L., JR. 86 E. Church St., Uniontown, Pa.
V. M. D., University of Pennsylvania, 1932
Vouchers: Samuel Glenn and Frank E. Lentz.

ENGE, EMERY H. Comfrey, Minn.
D. V. M., Iowa State College, 1932
Vouchers: O. C. Selby and D. T. Grady.

GUMP, R. H. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Kansas State College, 1933
Vouchers: C. H. Fauks and S. A. Watters.

HOPKINS, DAVID 21 Laurel St., Brattleboro, Vt.
D. V. M., Cornell University, 1930
Vouchers: G. N. Welch and L. H. Adams.

HOPSON, GEORGE H. Franklin Ave., Millbrook, N. Y.
D. V. M., Cornell University, 1928
Vouchers: W. E. Parks and F. D. Holford.

IVEY, WILLIAM E. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Kansas State College, 1934
Vouchers: C. H. Fauks and C. C. Hisel.

JACKSON, FAY F. Clear Lake, S. Dak.
D. V. M., McKillip Veterinary College, 1913
Vouchers: G. E. Melody and E. E. Flory.

KLEINFELD, ABRAHAM H. 411 36th St., Union City, N. J.
D. V. M., Cornell University, 1931
Vouchers: Alfred W. Meyer and James R. Kinney.

MCGEE, GEORGE M. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Alabama Polytechnic Institute, 1934
Vouchers: C. C. Hisel and L. J. Allen.

MCGEE, LUCIUS E. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Kansas State College, 1934
Vouchers: Edwin J. Frick and C. H. Fauks.

MITTEN, JAMES C. 313 Fifth Ave., N. W., Puyallup, Wash.
D. V. M., Terre Haute Veterinary College, 1916
Vouchers: C. E. Sawyer and C. M. Hamilton.

MORRISON, HAROLD L. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Iowa State College, 1931
Vouchers: C. C. Hisel and L. J. Allen.

NORDSTROM, E. HAROLD 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Iowa State College, 1929
Vouchers: C. C. Hisel and C. H. Fauks.

OELRICH, IVAN A. Chancellor, S. Dak.
D. V. M., Iowa State College, 1933
Vouchers: G. E. Melody and Marvin S. Thorpe.

PETERSON, ELLIOTT 529 W. Canadian St., Vinita, Okla.
D. V. M., Indiana Veterinary College, 1921
Vouchers: C. C. Hisel and L. J. Allen.

REAUGH, GEORGE T. 1650 Pattie Ave., Wichita, Kan.
D. V. M., Kansas State College, 1916
Vouchers: C. C. Hisel and L. J. Allen.

SMITH, ROY C. 421 S. Van Buren St., Enid, Okla.
D. V. M., Kansas City Veterinary College, 1914
Vouchers: C. H. Fauks and L. J. Allen.

WEAVER, CLARK A. Belleville, Ohio
D. V. M., Ohio State University, 1934
Vouchers: Maj. F. L. Holycross and W. F. Guard.

WILLERS, KARL H. 1028 Greenwood Ave., Canon City, Colo.
B. S., Colorado Agricultural College, 1931
D. V. M., Colorado Agricultural College, 1934
Vouchers: O. E. Robinson and E. W. Meads.

Applications Pending

SECOND LISTING

(See April, 1935, JOURNAL)

Boley, Loyd E., Burlingame, Kan.
Bostwick, David C., Box 922, Ardmore, Okla.
Danks, A. Gordon, Kansas State College, Manhattan, Kan.
Mahaffey, Claud O., Goltry, Okla.

Pearce, Grace, Marshall Browning Hospital Laboratory, Du Quoin, Ill.
Pearson, Charles C., 217 E. Sandusky St., Findlay, Ohio.
Thayer, Leslie F., c/o General Delivery, New Cumberland, W. Va.
Whitman, James R., c/o Dr. L. J. Allen, 334 Federal Bldg., Oklahoma City, Okla.

The amount which should accompany an application filed this month is $8.33 which covers membership fee and dues to January 1, 1936, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Connecticut Veterinary Medical Association. West Hartford, Conn. May 1, 1935. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. May 1, 1935. Dr. R. S. MacKellar, Jr., Secretary, 329 W. 12th St., New York, N. Y.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo., May 1, 1935. Dr. Harley B. Wood, Secretary, 2754 Meramec St., Saint Louis, Mo.

Houston Veterinary Association. Houston, Texas. May 2, 1935. Dr. T. A. Ward, Secretary, 2515 Ralph St., Houston, Texas.

New Mexico Veterinary Medical Association. Albuquerque, N. M. May 4, 1935. Dr. H. E. Kemper, Secretary, Box 464, Albuquerque, N. M.

Hudson Valley Veterinary Medical Society. U. S. Military Academy, West Point, N. Y. May 8, 1935. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. May 8, 1935. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.

Michigan-Ohio Veterinary Medical Association. Blissfield, Mich. May 9, 1935. Dr. E. C. W. Schubel, Secretary, Blissfield, Mich.

Tulsa County Veterinary Association. Tulsa, Okla. May 9, 1935. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Willamette Valley Veterinary Medical Association. Tillamook, Ore. May 11, 1935. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.

Ak-Sar-Ben Veterinary Medical Association. Elks Building, Omaha, Nebr. May 13, 1935. Dr. J. N. McIlnay, Secretary, 3251 Leavenworth St., Omaha, Nebr.

Chicago Veterinary Medical Association. Palmer House, Chicago, Ill. May 14, 1935. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

San Diego County Veterinary Medical Association. San Diego, Calif. May 14, 1935. Dr. L. K. Knighton, Secretary, 3438 Mountain View, San Diego, Calif.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. May 15, 1935. Dr. T. G. Beard, Secretary, 3684 Beverly Blvd., Los Angeles, Calif.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. May 21, 1935. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

Keystone Veterinary Medical Association. Philadelphia, Pa. May 22, 1935. Dr. C. S. Rockwell, Secretary, 5225 Spruce St., Philadelphia, Pa.

Colorado Veterinary Medical Association. Fort Collins, Colo. May 23-24, 1935. Dr. B. R. McCrory, Secretary, Colorado Agricultural College, Fort Collins, Colo.

Central New York Veterinary Medical Association. Constableville, N. Y. June 5, 1935. Dr. W. B. Switzer, Secretary, R. 5, Oswego, N. Y.

American Association of Medical Milk Commissions. Joint meeting with Certified Milk Producers' Association. Chalfonte Haddon Hall Hotel, Atlantic City, N. J. June 10-11, 1935. Dr. Harris Moak, Secretary, 360 Park Place, Brooklyn, N. Y.

American Society of Clinical Laboratory Technicians. Atlantic City, N. J. June 10-12, 1935. Miss Vivian Herrick, Secretary, Hillman Hospital, Birmingham, Ala.

New York State Veterinary Medical Society. Binghamton, N. Y. June 13-14, 1935. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

California State Veterinary Medical Association. San Diego, Calif. June 17-19, 1935. Dr. Cliff D. Carpenter, Secretary, 337 Central Ave., Los Angeles, Calif.

Eastern Iowa Veterinary Association, Inc. Mechanicsville, Iowa. June 18, 1935. Dr. John J. Strandberg, Secretary, 1005 8th Ave., Belle Plaine, Iowa.

North Dakota Veterinary Medical Association. North Dakota Agricultural College, Fargo, N. Dak. June 20-21, 1935. Dr. Lee M. Roderick, Secretary, North Dakota Agricultural College, State College Station, Fargo, N. Dak.

American Association for the Advancement of Science. Minneapolis, Minn. June 24-29, 1935. Dr. Henry B. Ward, Secretary, Smithsonian Institution Bldg., Washington, D. C.

Northwest Veterinary Medical Association. (Joint meeting of the Oregon, Washington and British Columbia Veterinary Medical Associations.) Victoria, B. C. July 8-10, 1935. Dr. W. Graham Gillam, Secretary, Cloverdale, B. C.

Maine Veterinary Medical Association. Bar Harbor, Maine. July 10, 1935. Dr. R. E. Libby, Secretary, Richmond, Maine.

Virginia State Veterinary Medical Association. (Joint meeting with the District of Columbia, Maryland and North Carolina Veterinary Medical Associations.) Richmond, Va. July 10-12, 1935. Dr. I. D. Wilson, Secretary, Virginia Polytechnic Institute, Blacksburg, Va.

Minnesota State Veterinary Medical Society and University of Minnesota Short Course for Veterinarians. University Farm, Saint Paul, Minn. July 11-12, 1935. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Missouri Veterinary Medical Association. Chamber of Commerce Bldg., Saint Joseph, Mo. July 16-17, 1935. Dr. Ashe Lockhart, Secretary, 800 Woodswether Rd., Kansas City, Mo.

American Veterinary Medical Association. Oklahoma City, Okla. August 27-30, 1935. Dr. H. Preston Hoskins, Secretary, 221 N. La Salle St., Chicago, Ill.

STATE BOARD EXAMINATIONS

Kansas State Board of Veterinary Examiners. Veterinary Building, Kansas State College, Manhattan, Kan. May 24, 1935. Dr. Thomas P. Crispell, Secretary, Parsons, Kan.

Nebraska Bureau of Examining Boards. State House, Lincoln, Neb. June 24-25, 1935. Applications must be on file at the Bureau not later than 15 days prior to date of examination. Mrs. Clark Perkins, Director, Bureau of Examining Boards, State House, Lincoln, Neb.

Massachusetts Board of Registration in Veterinary Medicine. Department of Civil Service and Registration, Boston, Mass. July 9-10, 1935. Applications may be obtained from the Secretary, Dr. E. W. Babson, Gloucester, Mass.

Nature fits all her children with something to do.—LOWELL.

STUDIES OF TOTAL ERYTHROCYTE AND LEUKOCYTE COUNTS OF FOWLS

II. Effect of 48-Hour Starvation on Total Erythrocyte and Leukocyte Counts*

By E. IRENE PALMER *and* JACOB BIELY
University of British Columbia, Vancouver, B. C.

One of the first symptoms shown by many diseased birds is lack of food consumption. Such birds, when submitted for postmortem examination, are found as a rule to have empty crops, which indicates that they may have been without food from 24 to 48 hours. In fact, if paralyzed, and more particularly if in advanced stages of paralysis, birds may be actually starved through inability to reach and consume food. These observations suggested a study of the effect of moderate starvation on the total erythrocyte and leukocyte counts of normal fowls.

MATERIAL AND METHODS

The methods followed in counting the red and white blood-cells were the same as those reported in a previous paper.[1] The birds were raised and kept in battery brooders, receiving mash and water as their only food. Total erythocyte and leukocyte counts were made of each bird before it was starved. At the time that blood was taken for counting, the crop of each bird contained some food. Immediately after a sample of blood was taken, the bird was placed in a cage and both food and water withheld for 48 hours. Blood counts were then made approximately on the hour corresponding to that at which the count was made previous to starvation.

DATA

Data were obtained on 39 birds. Table I shows the total erythrocyte and leukocyte counts of a group of birds that were counted once previous to and once following a 48-hour starvation period.

Table II shows the average of duplicate counts obtained with birds whose blood-cells were counted twice before and twice subsequent to starvation.

Table III shows the results obtained with five birds that were counted on each of four successive days previous to starvation.

*Received for publication, November 12, 1934.

Only the average of the four counts on each bird is shown in the table. The birds were then starved 48 hours and total red and white counts were made. The birds then were given mash and water and counts were made 24 hours after feeding.

On examining tables I, II and III it will be seen that 31 out of 39 birds showed an increase in the erythrocyte counts following starvation, while eight birds showed a slight decrease.

The results of the leukocyte counts (tables I, II and III) show that, with one exception, a consistent decrease of leukocytes occurred in all the birds. The decrease in the three groups of birds reported was 23.1, 27.7 and 28.3 per cent, respectively. A statistical analysis of the combined groups 1, 2 and 3 shows that the decrease in the leukocyte counts previous and subsequent to starvation is statistically significant.

TABLE I—*Erythrocyte and leukocyte counts of fowls before and after starvation for 48 hours (single counts).*

BIRD	WEIGHT BEFORE STARVING (GM)	DECREASE IN WEIGHT AFTER STARVING (GM)	ERYTHROCYTE COUNT AFTER STARVING*	INCREASE OR DECREASE IN ERYTHROCYTE COUNT AFTER STARVING*	LEUKOCYTE COUNT BEFORE STARVING	INCREASE IN LEUKOCYTE COUNT AFTER STARVING
1	1340	185	3050	− 70	43,340	11,120
2	1395	185	2700	+ 180	43,940	16,330
3	1395	235	2690	+ 545	30,885	6,385
4	1350	195	2745	+ 850	45,220	5,835
5	1535	205	2580	+1135	53,555	15,815
6	1555	202	2865	− 355	34,775	9,445
7	1645	135	2585	+ 140	36,610	11,170
8	2165	230	2530	− 95	51,720	16,020
9	1520	155	2375	+ 425	31,665	11,665
10	1383	114	2555	+ 215	74,770	19,670
11	1489	132	2605	+ 525	64,770	9,890
12	1467	147	2755	+ 145	54,000	6,230
13	1444	144	2855	+ 160	77,770	13,550
14	1688	171	2775	− 60	47,880	4,550
15	1418	104	2665	+ 350	97,880	34,880
16	1669	240	2940	− 340	42,660	7,330
17	1311	92	2135	+ 925	43,000	4,000
18	1300	128	2955	+ 330	40,770	8,890
19	1567	212	2605	+ 230	53,220	11,000
20	1455	140	2715	− 625	36,775	8,325
Average	1504.5	167.5	2684	+ 230	50,260	11,605
Perctg.		11.1		+ 8.5		23.1

*Expressed in thousands.

Table III shows the results of counts that were made 24 hours after the birds were fed following the starvation period. It will be seen that the erythrocyte and leukocyte counts were comparable to the average counts previous to starvation.

Discussion

The 48-hour period of starvation was chosen in preference to a shorter period, in order to avoid the immediate effects of so-called digestion leukocytosis.

According to Burnett,[2] the investigations of Pohl, Rieder and Goodal, Gulland and Paton, on dogs, and of Brinckerhoff and Tyzzer, on rabbits, have shown that there is a diminution in the number of leukocytes after a fast of twelve hours or longer and that there is an increase after a fasting dog or rabbit is fed. Brinckerhoff and Tyzzer found that in rabbits a fast of twelve hours or longer caused a decrease in the leukocytes averaging approximately one-third the initial count. Feeding fast-

Table II—*Erythrocyte and leukocyte counts of fowls before and after starvation for 48 hours (duplicate counts).*

Bird	Weight Before Starving (Gm)	Decrease in Weight After Starving (Gm)	Erythrocyte Count After Starving*	Increase or Decrease in Erythrocyte Count After Starving*	Leukocyte Count Before Starving	Increase in Leukocyte Count After Starving
21	1445	150	2805	+ 15	53,553	17,553
22	1410	125	2817	+ 753	45,330	11,553
23	1890	90	2330	+ 370	42,050	11,500
24	1725	200	2055	+ 467	34,385	9,560
25	1975	250	2635	+ 420	44,550	10,165
26	1430	230	2730	+ 687	32,000	6,168
27	1665	288	2365	+ 320	46,440	9,415
28	1385	207	2560	+ 95	36,385	7,835
29	1370	60	2135	+ 677	56,805	21,055
30	1965	240	2447	+ 103	39,440	9,890
31	1550	225	2760	+ 120	31,385	7,615
32	1745	385	2720	+ 610	19,055	+385
33	2000	390	2897	+ 437	60,500	24,505
34	1575	115	2770	+ 50	33,110	13,110
Average	1651	211	2573	+ 366	41,070	11,396
Perctg.		12.7		+14.2		27.7

*Expressed in thousands.

TABLE III—*Erythrocyte and leukocyte counts of fowls before (average of four counts) and after starvation for 48 hours (one count) and 24 hours after feeding (one count).*

BIRD	WEIGHT BEFORE STARVING (GM)	DECREASE IN WEIGHT AFTER STARVING (GM)	ERYTHROCYTE COUNT BEFORE STARVING*	INCREASE OR DECREASE IN ERYTHROCYTE COUNT AFTER STARVING	LEUKOCYTE COUNT BEFORE STARVING	DECREASE IN LEUKOCYTE COUNT AFTER STARVING	ERYTHROCYTE COUNT 24 HOURS AFTER FEEDING	LEUKOCYTE COUNT 24 HOURS AFTER FEEDING
1	1267	84	2604	+ 96,000	45,690	10,810	2850	41,110
2	1385	120	2931	+509,000	33,882	8,662	2905	37,330
3	1557	151	3053	−273,000	42,830	8,830	2840	43,880
4	1274	132	2612	−317,000	44,995	8,555	2830	40,110
5	1551	166	2705	+105,000	78,662	32,782	2535	67,550
Average....	1407	131	2781	+ 24,000	49,211	13,928	2792	45,996
Percentage.		9 3		+ .86		28 3		

*Expressed in thousands.

ing rabbits caused an increase two to six hours after the beginning of feeding.

In a recent paper, Cole[3] reported that the leukocyte counts of pigs that had just been fed were 29 per cent higher than those of pigs that had not been fed for twelve hours. Our data show that, in three lots of birds reported in this investigation, the leukocyte counts decreased 23.1, 27.7 and 28.3 per cent respectively after 48 hours of starvation.

No attempt was made to determine the effect of various kinds of feed (mash or grain) on the total erythocyte or leukocyte counts previous and subsequent to starvation. According to Burnett, the kind of food seems to exert a considerable influence, a protein (meat) diet showing the greatest effect and a carbohydrate or fat diet little or no effect. This subject would seem to warrant further consideration.

Most investigators have found that there is a wide variation in the number of erythrocytes and leukocytes in the blood of normal fowls, but few data regarding the cause of such variation are available. A systematic study of the blood of fowls, with reference to the time of feeding, type of food, and other factors, may throw some light on the apparent fluctuations of total red and leukocyte counts of normal individuals and furthermore on the variations reported by different investigators.

It would seem that in making counts of normal or diseased fowl, consideration should be given to the time interval between the last feeding and the actual time of counting. A standard procedure in this regard would appear to be essential in order to avoid an obvious source of fluctuation in erythrocyte and leukocyte counts.

SUMMARY

(1) Erythrocyte and leukocyte counts were made of three groups of birds previous and subsequent to 48 hours of starvation.

(2) In 31 out of 39 birds, an increase in the erythrocyte counts was found, while in eight there was a decrease.

(3) With one exception, all birds showed a decrease in the leukocyte counts. The percentage decrease for the three groups was 23.1, 27.7 and 28.3 respectively.

(4) It is concluded from the data that, in making blood counts, the time interval between the last feeding and the time the counts are made should be taken into consideration. To avoid an obvious source of fluctuation, resulting from partial

starvation, a standard procedure with regard to time of feeding and making erythrocyte and leukocyte counts should be followed in the study of normal or diseased fowls.

Note: Due to lack of funds this phase of the study on "Total Erythrocyte and Leukocyte Counts of the Domestic Fowl" has not been completed. The data presented in this paper, as far as they go, are of interest and may stimulate further research along the lines indicated.

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President's Train Wrecks Veterinarian's Auto

The automobile of Dr. L. J. Herring (K. C. V. C. '09), of Wilson, N. C., was demolished by the special train bearing President Roosevelt from a fishing trip in Florida to New York. The car, which had been stolen from its parking-place while Dr. Herring was at a theater, had been abandoned by the thieves at a grade crossing near Wilson. The special train was delayed 15 minutes. Two railroad detectives who were on the presidential train remained overnight to make an investigation.

Illinois Sales Tax Ruling for Veterinarians

How the Illinois retailers' occupation tax (sales tax) works with reference to veterinarians and their services is set forth in rule 79, of the Illinois Department of Finance. It reads as follows:

> Veterinarians are primarily engaged in the business of rendering professional services to the owners of domestic animals, through care, medication and treatment of such animals. They are users and consumers of all such items of tangible personal property as drugs, medicines, bandages and dressings, serums, tonics, and the like which are used by them in connection with the performance of such professional services. Persons selling such items to veterinarians for use in the performance of professional services become liable for Retailers' Occupation Tax. For instance, sales of hog serums to veterinarians for use in the performance of vaccination services constitute sales to users or consumers.
>
> Where veterinarians engage in the business of selling any tangible personal property, such as live stock tonics or remedies, to users or consumers, separately and apart from the rendering of professional services, they become liable for the tax.

Thus a veterinarian is exempt from the tax, as long as he renders professional services only.

SIGNIFICANCE OF STREPTOCOCCI, AND A HIGH LEUKOCYTE COUNT, H-ION CONCENTRATION AND CHLORINE CONTENT IN COW'S MILK*

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A number of reports have been published of the finding of streptococci similar to the type associated with mastitis (*Streptococcus agalactiae, S. mastitidis)* in milk from cows with apparently normal udders, but the information given as to how the condition of the udders was determined is in most cases vague and indefinite. As the clinical symptoms of the form of mastitis which generally affects cows in milk (catarrhal mastitis) are so slight that they could easily be overlooked in a casual examination, especially in the beginning of the disease, it would seem that a careful clinical examination of the cow is one of the essentials of such an investigation. A knowledge of the previous and subsequent history of the cow in regard to mastitis would also be of value. Many of the published reports of studies made to determine the leukocyte count, chlorine content, reaction, and similar properties of normal milk are also incomplete in this respect and are likewise lacking in information regarding the stage of lactation and age. In a number of investigations, however, these elements of the problem have received attention and the results obtained are of considerable interest in connection with the question of whether mastitis streptococci may be present in the udder of the cow without causing mastitis.

The milk of two cows was found by Hastings and Hoffmann[1] to contain streptococci apparently of the mastitis type; that of one cow over a period of nine months and of the other over a period of 15 months. During the entire time, the milk of both was of normal appearance while the leukocyte count varied from 122,000 to nearly 2,000,000. The udder of one cow was normal on physical examination; that of the other had a slight induration in one quarter.

Examining the milk of 50 cows in one stable, Jones[2, 3] found 16 (32 per cent) infected with streptococci of the mastitis type. The milk was normal in appearance. Five of the infected cows

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had had mastitis during the current lactation period, three developed mastitis during the four months the cows were under observation, but the eight others showed no signs of the disease.

Steck[4-6] made extensive studies of the bacteria present in udders in which no abnormal condition could be detected by palpation and which secreted milk of normal appearance and taste. In the milk drawn from such udders with precautions against external contamination, he found streptococci of the mastitis type, also *Streptococcus lacticus*, micrococci, *Brucella abortus* Bang, and diphtheroid forms *(Corynebacterium bovis)*. Usually, one species predominated, generally the micrococci, while the total number of organisms and the predominating species in the milk samples from the same quarter generally continued the same for months. All of these organisms, he found, caused some increase in the number of leukocytes in the milk, the degree depending upon the number of organisms present, but the mastitis streptococci were more active in this respect than the others. The reaction of the udder to this "normal" or "latent infection" did not differ from that which occurs in clinical mastitis except in degree. On the other hand, Minett, Stableforth and Edwards,[7] while recognizing that mastitis streptococci may be present in milk from udders in which there is "no obvious pathological change" and even when laboratory examination of the milk may fail to reveal "distinct abnormality," are not willing to admit that these organisms are normal inhabitants of the udder because "in most quarters in which they can be demonstrated pathological disturbance arises sooner or later."

Seelemann[8] asserts that when mastitis streptococci are present changes can be demonstrated in the milk. These changes may be slight, however, and may not be apparent unless repeated examinations are made of samples from infected and non-infected quarters and the results compared. He uses the term "latent infection" for this condition. When a herd is infected "to a medium or to a considerable degree," he finds that sooner or later the greater proportion of the cows become infected. External evidence of mastitis may be apparent to the owner or attendants, or it may exist for a long time without being observed by them. When physical symptoms appear, latent infection has become manifest, due to the operation of certain influences. These influences, foremost among which is incomplete milking, play a more important rôle in the so-called "outbreaks" of the disease than the spread of infection. An "outbreak" is only a manifestation of a previously existing latent infection.

Milk samples from 439 udder quarters "showing no clinical evidence of mastitis" were examined by Hucker, Trudell and Jennings[9] for mastitis streptococci and 48 per cent were found to be infected. In a study of the udders and milk of 70 cows, Rosell and Miller[10] found streptococci of the mastitis type present in 94 (41 per cent) of the quarters which reacted to the leukocyte, pH, chlorine or catalase test. They were also present in 10 (21 per cent) of the 48 quarters which did not react to any of these tests. The milk was abnormal in appearance from only 15 of the infected quarters.

Plan Of Investigation

Two groups of cows were selected for this investigation—one of 22 cows (group A) in a herd of 90 pure-bred Holsteins and one of 24 (group B) in a herd of 80 pure-bred Ayrshires, 46 in all. The selections were made entirely on the basis of age and stage of lactation in order that different periods of lactation (age) and various stages of lactation would be represented. The cows in each group were stabled in adjoining stalls in the same stable with other cows in the herd to which they belonged and were milked before the other cows. Seven in group B were milked by machine. The other cows in this group and all those in group A were milked by hand. The usual dairy ration was fed, the quantity of concentrates being determined by the quantity of milk produced. The sanitary conditions and methods of milking were such that the plate count of the milk of each herd was usually not above 10,000. Both herds were accredited as free from tuberculosis and Bang's disease. At each milking, the first three or four streams from each quarter were drawn on a fine copper-wire strainer by the milker and examined for flakes, of which a report was made if any were observed.

A physical examination of the udder of each cow was made every four weeks and a sample of milk was drawn from each quarter at the same time. Five or six cows were examined at a time so that each group was visited once each week, affording an opportunity to note any unusual conditions.

Ordinary 30-cc culture tubes with rubber stoppers were used to collect the milk samples, after being sterilized and marked in the laboratory. After the udder was brushed as clean as possible, it was wiped with a damp cloth. Three or four expressions of milk were drawn on the wire strainer and examined for flakes, and the sample for examination was then drawn into the culture tube, the latter being held as nearly horizontal as possible to avoid contamination. The cow was milked dry by the regular

milker, after which the udder was carefully examined by inspection and palpation. The milk samples were then taken to the laboratory, having in the meantime been kept in a cool place out of the sun but not iced.

Upon arrival at the laboratory one to two hours later, the samples from the cows in group A were immediately plated, tested for pH and chlorine content, the leukocyte count being made the next morning; while the samples from the cows in group B were placed in the ice-box until the following morning and then examined. The methods of examination used were the following:

Plate method: One cc of the milk, after thorough shaking, was diluted with 99 cc of sterile distilled water. One cc of this dilution then was transferred to a Petri dish and thoroughly mixed with 10 to 12 cc of Difco veal infusion agar to which 1 cc of citrated horse blood had been added after the agar had been cooled to 45°C. The plates, after hardening, were placed in the incubator at 38°C. and observations were made at 24 and 48 hours. At the end of 48 hours, the colonies were counted and subcultured in 10 cc of veal infusion to make sure of their morphology, as only streptococcus colonies were considered. Cultures were made also for classification, using litmus milk, methylene blue milk, and various sugars, including dextrose, saccharose, lactose, salicin, raffinose, mannite and insulin.

pH value: The La Motte method, after Brown, was adopted to determine the H-ion concentration of the milk. This method requires a very small amount of milk and other material. One loopful of well-shaken milk was added to nine drops of distilled water in each cell, followed by one drop of bromthymol blue.

Chlorine content: As simplicity was desired, the chlorine test No. 3, described by Hayden,[11] was used. To 5 cc of a 1.3415 silver nitrate solution in small test-tubes, 2 drops of a 10 per cent potassium chromate solution was added followed by 1 cc of the milk to be tested. The tubes were not shaken, however, as recommended by Hayden, as it was found by comparing the results with those obtained by titration that a more conservative and closer chlorine valuation was obtained in this way. A positive reaction to this test indicates that the milk contained 0.14 per cent or more of chlorine. When practically the whole tube showed a brick-red color, the reaction was considered negative; when slightly more than half of the tube showed yellow, slightly positive; and when the whole tube turned yellow, with only a slight brick-red band at the top, within 1 to 5 minutes, positive.

Leukocyte count: The Stewart-Slack method, as described in the Standard Methods of Milk Analysis, fifth edition, published

by the American Public Health Association, was used to determine the leukocyte count. Into especially constructed glass tubes, closed at both ends with rubber stoppers, 2 cc of milk is pipetted. The tube is then centrifugalized at 2,500 to 3,000 r.p.m. for ten minutes and after the cream and milk layers are removed, the sediment attached to the remaining rubber stopper is smeared over 4 sq cm on a glass slide. After staining with methylene blue, the leukocytes in 10 to 20 fields are counted and the average count multiplied by 6,000 to determine the number of leukocytes in 1 cc of milk.

Discussion of Results

The udders of 26 (56.5 per cent) of the 46 cows studied in this investigation were found to be infected with streptococci similar to the mastitis type in one or more quarters. In all, 75 quarters were found to be infected, or 40.7 per cent of the total number studied (table I). Forty-nine quarters were infected originally and the infection extended to 26 additional quarters in the same cows during the period covered by the investigation (table II). It is possible, of course, that some of the quarters found infected secondarily may have been infected when the original infection was found, as a single negative culture test does not exclude the possibility of streptococci being present. The infection was less extensive in group A than in group B, there being 12 cows with 30 quarters infected in the former and 14 cows with 45 quarters infected in the latter, although sanitary conditions, methods of

Table I—*Incidence of streptococcic infection in different periods of lactation.*

Period of Lactation	Cows	Cows Infected with Streptococci		Quarters Infected with Streptococci	
		No.	%	No.	%
1st	13	2	15.3	5	62.5
2nd	8	3	37.5	10	83.3
3rd	5	3	60.0	5	41.6
4th	6	4	66.6	12	75.0
5th	5	5	100.0	14	70.0
6th	3	3	100.0	10	83.3
7th	3	3	100.0	10	83.3
8th	2	2	100.0	5	62.5
9th	0	0	0.0	0	0.0
10th	1	1	100.0	4	100.0
Totals....	46	26	56.5	75	40.7

milking, feeding and herd management were approximately the same.

TABLE II—*Quarters infected with streptococci and results of milk tests.*

CONDITION OF QUARTERS	TOTAL QUARTERS	LEUKOCYTES (1,000,000 OR OVER)		pH (ABOVE 6.8)		CHLORINE (0.14 OR OVER)	
		No.	%	No.	%	No.	%
Originally Infected	49	25	51.0	28	57.1	31	63.2
Secondarily infected	26	8	30.7	8	30.7	12	46.1
Totals	75	33	40.0	36	48.0	43	57.3

The streptococcic infection persisted in most of the quarters in which it appeared. Three cows with eight infected quarters were "dried off" and when they "freshened," eight to 16 weeks later, all but one of the quarters were still infected. Cow 815, with all four quarters infected, came in fresh with one quarter free of streptococci. These observations are in agreement with the experience of Steck,[4-6] who found that when mastitis streptococci become established in the udder they usually remain for months, even through several lactation periods. Seelemann[8] also noted that the infection is likely to continue. The tendency of *Br. abortus* Bang to persist in the udder of an infected cow is well known. It may be, as O. Bang, Bendixen and Orskov[12] state, that the udder is less able than other organs to free itself of infection, although it has the power to inhibit bacterial growth (Jones,[2,3] Steck[4-6]).

The incidence of streptococcic infection in the cows studied increased with each successive period of lactation (table I). This is in agreement with Seelemann's[8] observation that the incidence of mastitis increases with the age of the cow. There was also a general tendency for a greater number of quarters to be infected in the older cows (table I).

The results obtained with the other methods of examination at the time the streptococci were first observed in the milk from the infected quarters are shown in table II, the data being presented in a form to indicate the number and percentage of quarters from which milk was obtained containing a million or more leukocytes per cc, a pH above 6.8, or a chlorine content of 0.14 or over, these being the generally accepted limits for normal milk. The results of the tests made when the streptococci were dis-

covered are considered, instead of those of subsequent tests, because this is what would ordinarily happen in practice.

As the data in table II show, the milk from 25 (51 per cent) of the 49 quarters infected originally, had a leukocyte count of 1,000,000 or over; that from 28 (57 per cent) of these quarters had a pH above 6.8, while the milk from 31 (63 per cent) had a chlorine content of 0.14 or over. It will be noted that the milk from a considerable proportion of the quarters originally infected was not positive to these tests. At the subsequent periods of examination, the milk from some of these negative quarters gave positive results, while that from some of the quarters that were positive at first had a leukocyte count, pH, and chlorine content that was within the normal limits. There was no correlation between these changes and the number of streptococci present.

The leukocyte count of the milk from 8 (30.7 per cent) of the 26 quarters infected secondarily was a million or over at the time the streptococci were first observed and the milk of an equal number of these quarters had a pH above 6.8, while the chlorine content was 0.14 or over in the milk of 12 (46 per cent), the percentage of positive results in each instance being lower than for the milk from the quarters infected originally; indicating that the udder had acquired an increased resistance or tolerance for the streptococci. This is in line with the experience of Jones,[2,3] Steck[4-6] and others.

Using the cow as the unit of classification, it is found that in 23 (83.3 per cent) of the infected cows, the milk of one or more of the quarters infected originally had a leukocyte count, a pH, or a chlorine content in excess of the normal limits at the time the streptococci were first observed. Considering the results of the three tests individually, 18 cows were positive to the leukocyte test and 21 were positive to each of the two other tests but not all the same cows to each test. Thirteen of the infected cows, with 25 quarters infected, gave milk from 18 of these quarters which was positive to all three tests, while the milk from three cows (219, P3 and 64), with three infected quarters, was negative to all three tests. Some of the subsequent samples of milk from these quarters reacted to one or the other test but most of them were negative. Judging the 26 infected cows entirely on the results of any one of the three tests, 23 would be classed as abnormal; on the basis of the leukocyte test alone, 18, and on the basis of the pH or chlorine tests, 21. It cannot be said positively how many of these cows would have been classed as abnormal if composite samples consisting of an approximately equal quantity of milk from each quarter had been examined instead

of individual samples from each quarter, but it is very likely that the number would have been smaller.

Three of the cows reacting to all three tests (0088, 440 and 390) showed clinical symptoms of mastitis. The record of these cows follows:

Cow 0088: The right hind quarter of the udder was somewhat atrophied and had a small indurated area at the time of the first examination, which was made in the ninth week of the eighth lactation period. Flakes were observed in the first few expressions of milk from this quarter and the quantity secreted was much less than that produced by any of the three other quarters. No streptococcic colonies developed on the blood-agar plates inoculated with milk from this quarter but the pH and chlorine content exceeded the normal limits and the leukocytes were too numerous to count. At the succeeding examination, four weeks later, the milk contained streptococci (50,000 per cc) and the pH, chlorine content, and leukocyte count were again above the normal limits. Flakes were constantly present in the milk from this quarter. These conditions continued without change during the succeeding 28 weeks the cow was under observation except that the pH of the milk was normal at the last two examinations and the cell count was down to 105,000 at the last examination. Small indurated areas were present in the three other quarters but no streptococci were found in the milk from these quarters at any time, and cell count, pH, and chlorine content were always within the normal limits. This was a case of catarrhal mastitis which had evidently started some time before the first examination was made.

Cow 440: The first time this cow was examined the left front quarter was somewhat atrophied and contained a small indurated area, while the secretion had decreased to 30 to 50 cc at a milking, although the animal was then in the 17th week of the first lactation. Streptococci were present in the milk in considerable number, the colonies on the blood-agar plates being too numerout to count, and the leukocyte count, pH, and chlorine content were high. The milk was in this condition from the first to the seventh examination period (41st week of lactation), when the cow was dried off. Flakes were observed repeatedly but otherwise the secretion from this quarter was normal in appearance most of the time, but sometimes became "thick." A small indurated area also was found in the left hind quarter at the first examination, and a few small flakes were observed in the first few streams of milk from the right hind quarter at the time of the

fifth examination period but the milk from these two quarters and the right front quarter did not at any time show evidence of streptococcic infection or any abnormal results to the other tests except that at the last examination the leukocyte count of the milk from the right front quarter was over a million. The cow was then in the 41st week of lactation. This case was complicated with a traumatism, the left front quarter having been cut with a hoe used to clean the stable floor. The accident occurred before the cow came under our observation and it was not possible to determine to what extent the course of the streptococcic infection was affected by the injury.

Cow 390: This cow was examined in the 40th and 44th weeks of the fifth lactation. Nothing abnormal was found in the udder and the milk was of normal appearance except for a few small flakes in the first streams from the right hind quarter, but the pH and chlorine were high in the milk of all four quarters at the first examination and that from the left front quarter had a leukocyte count of 1,200,000. On the second examination, the udder was normal and the milk was normal in appearance. The pH was still high in the milk from the left front quarter but was just within the normal limit in the milk of the three other quarters. Chlorine was still high in the milk of all four quarters but the leukocyte count was within the normal limits. No streptococci were found at either examination in the milk of any of the quarters. After a "dry" period of two to three months, the cow calved. The animal was examined on the fourth day after calving. The left front quarter was then swollen but was not hot or painful; the secretion consisted entirely of a thick, yellow, viscid fluid, and innumerable colonies of streptococci appeared on the blood-agar plates inoculated with it. The pH was rather low (6.4) but the chlorine content was above normal and the cells were too numerous to count.

The other quarters were normal on physical examination and the milk was normal in appearance but chlorine exceeded the normal limit in the milk from the right front quarter. No evidence of streptococci in these quarters appeared on the blood-agar plates and the leukocyte count was not above the normal limit. A week later, the swelling had disappeared from the left front quarter but there was an indurated area the size of an egg in the region where the large milk-canals join the milk-cistern. The secretion had improved in appearance and contained fewer streptococci (75,000 per cc) but the pH, chlorine and leukocyte count were high. Streptococci also were present at this time in the right front and right hind quarters but the milk from these

and from the left hind quarter was normal in pH, chlorine and cell count. A month later, the left hind quarter also was infected but the milk did not show any increase in pH, chlorine or leukocytes. The left front quarter gradually decreased in size and there was also a decrease in the secretion. Flakes were repeatedly present in the first three or four streams from this quarter but otherwise the milk was of normal appearance.

At the last two examination periods, 14 and 18 weeks after calving, there was no evidence of the presence of streptococci in the milk of any of the quarters. The milk from the left front quarter continued high in pH, chlorine and cell count but the milk from the other quarters was normal to these tests. This case was of a more acute type and the change in the appearance of the secretion of the affected quarter was more pronounced in the beginning than is ordinarily observed in catarrhal mastitis. These differences may have been due to the cow being in the colostral period when the attack began. Others have observed that the mastitis streptococci are likely to produce an acute type of mastitis in the "fresh" cow and a subacute or chronic type later in lactation (Kitt,[13] De Bruin, cited by Ernst[14]).

The 20 other reacting cows showed nothing unusual except the appearance, temporarily and infrequently, of a few small flakes in the milk, and the presence of limited areas of induration in the udders. Flakes were observed in the milk of ten of these cows. The flakes were very small, few in number, and were present in only the first few streams of milk, usually for only a single milking. They appeared in the milk of 17 quarters at various times: One quarter in four cows, two quarters in five cows, and three in one cow. Three of these quarters were not infected with streptococci. The flakes appeared in the milk of twelve quarters only once; in the milk of four quarters twice, one a non-infected quarter, and in the milk of one quarter four times, also a non-infected quarter.

One of the three infected cows which were normal to all three of the tests had flakes once in the milk of one quarter, making a total of eleven cows and 18 quarters showing flakes, not including the three clinical cases, or 47.8 per cent of the infected cows (excluding the clinical cases) and 26 per cent of the quarters. Flakes similar in appearance to these are one of the earliest clinical symptoms of catarrhal mastitis, but when the disease develops they are followed or accompanied by other visible changes in the milk and usually by certain changes in the udder which can be detected by physical examination. None of these changes occurred in any of these cows during the time they were

under observation, except that small areas of induration appeared in five quarters after they had become infected with streptococci. One cow had two such quarters and three others one each. Indurated areas were present in other infected quarters but they were present before the infection.

Similar flakes were observed in the milk of nine quarters in seven of the non-infected cows. One quarter showed flakes twice, the others only once. The incidence of flakes was somewhat lower, however, in the non-infected than in the infected cows, 35 per cent of the non-infected cows and 11.2 per cent of the quarters showing this change (table VI). The milk from these quarters did not show as extensive changes in the leukocyte count as that from similar quarters in the infected cows. Only 12 per cent of the milk samples from the non-infected quarters had a leukocyte count of a million or over, compared with 66.6 per cent from the infected quarters. All of the samples with the high cell count from the non-infected cows had a pH and a chlorine content above normal but only 56.2 per cent of those from the infected cows had a pH above the normal limit, while the chlorine was high in 68.7 per cent (table III).

Areas of induration were found in the udders of 23 of the 26 cows infected with streptococci; in 43 (57.3 per cent) of the infected quarters and 16 (55.1 per cent) of the non-infected quarters (tables IV and V). The 23 cows showing this condition included 17 of those that were positive to one or the other of the milk tests but which did not develop definite clinical symptoms, the three cows that were negative to all three tests, and the three clinical cases. Three of the reacting cows (247, P25 and 513) did not show any induration. The indurated areas varied in size from that of a pea to a walnut, most of them being nearer the size of the former, and the greater portion were located in the region where the large milk-canals join the milk-cistern. All of them persisted throughout the period of investigation, indicating that they were of a fibrotic character, but none of them increased in size to any perceptible degree while the cows were under observation, which would seem to justify the assumption that the condition was not progressive. In 21 of the infected quarters they were present before streptococci were found in the milk.

Similar areas were present in 16 quarters in eleven of the non-infected cows, or 20 per cent of the quarters and 55 per cent of the cows, a smaller proportion than for the infected cows (table V). The cell count exceeded a million in the milk of one of these quarters (6.2 per cent); the pH was above 6.8 in that of three (18.7 per cent) and the chlorine was 0.14 or more in that of eight

TABLE III—*Leukocyte count, pH and chlorine content of milk showing flakes.*

Leukocyte Count	Samples from Non-Infected Quarters						Samples from Infected Quarters					
	Flakes		pH (Above 6.8)		Chlorine (0.14 or More)		Flakes		pH (Above 6.8)		Chlorine (0.14 or More)	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
1,000,000 or over	3	12.0	3	100.0	3	100.0	16	66.6	9	56.2	11	68.7
500,000 to 1,000,000	2	8.0	1	50.0	0	0.0	2	8.3	1	50.0	2	100.0
250,000 to 500,000	3	12.0	1	33.3	3	100.0	2	8.3	1	50.0	1	50.0
100,000 to 250,000	5	20.0	1	20.0	2	40.0	3	12.5	0	0.0	0	0.0
100,000 or less	12	48.0	0	0.0	0	0.0	1	4.1	1	100.0	1	100.0
Totals	25		6	24.0	8	32.0	24		12	50.0	15	62.5

TABLE IV—*Incidence of induration in different periods of lactation (cows infected with streptococci).*

PERIOD OF LACTATION	COWS	INFECTED QUARTERS	COWS WITH INDURATED QUARTERS		INFECTED QUARTERS INDURATED		NON-INFECTED QUARTERS INDURATED	
			No.	%	No.	%	No.	%
1st	2	5	2	100.0	4	80.0	1	33.3
2nd	3	10	2	66.6	3	30.0	1	50.0
3rd	3	5	2	66.6	3	60.0	3	42.8
4th	4	12	4	100.0	10	83.3	2	50.0
5th	5	14	5	100.0	8	57.1	3	50.0
6th	3	10	3	100.0	4	40.0	2	100.0
7th	3	10	3	100.0	9	90.0	1	50.0
8th	2	5	2	100.0	2	40.0	3	100.0
10th	1	4	0	0.0				
Totals	26	75	23	88.4	43	57.3	16	55.1

(50 per cent). With the infected cows the incidence of induration increased with age but as most of the non-infected cows were in the earlier periods of lactation the data on these are inconclusive in this respect. This difference must also be considered in comparing the incidence of induration in the two classes.

On the basis of the conditions found, the 26 infected cows may be divided into two groups, as follows:

1. The three cows that showed definite clinical symptoms of mastitis.

2. The 23 cows that did not show any visible changes in the milk except a few small flakes temporarily and infrequently, and

TABLE V—*Incidence of induration in different periods of lactation (cows free from streptococci).*

PERIOD OF LACTATION	COWS	COWS WITH INDURATED QUARTERS		INDURATED QUARTERS	
		No.	%	No.	%
1st	11	7	63.6	11	25.0
2nd	5	1	20.0	1	5.0
3rd	2	2	100.0	3	37.5
4th	2	1	50.0	1	12.5
5th	..	..	.....	..	.....
6th	..	..	.....	..	.....
7th	..	..	.....	..	.....
8th	..	..	.....	..	.....
10th	..	..	.....	..	.....
Totals	20	11	55.0	16	20.0

in the udders of which no change could be found on physical examination except the small areas of induration detected in 20. The milk of 20 of these 23 cows, including the three that did not have indurated areas in the udder, had a leukocyte count, a pH, or a chlorine content in excess of the normal limits at the time the streptococci were first observed. Flakes and indurated areas and milk changes of a similar character were observed in some of the non-infected cows but not to the same extent.

According to Steck,[4-6] when streptococci are present in the udder and the milk has a high cell count but is not visibly changed, there is a mild catarrhal condition affecting the mucous membranes of the udder. A condition of this kind might be present without causing any change in the udder that could be detected by physical examination. We had the opportunity of making a postmortem examination of the udders of two of the cows giving milk of normal appearance with a leukocyte count of over 1,000,000 but found no congestion or inflammation or epithelial defects in the mucous membrane of the milk-cistern nor in the mucous membrane of the milk-canals as far as they could be traced. These parts are usually affected in catarrhal mastitis (Frei[15]). A macroscopic examination cannot be depended upon, however, to find any or every alteration which might increase the leukocytes, chlorine, or pH of the milk. In udders infected with *Br. abortus*, in which no alterations were apparent on macroscopic examination, lesions which accounted for the high cell content of the milk were found on histological examination (Runnells and Huddleson,[16] Hallman, Sholl and Delez[17]). There is no reason to suspect that *Brucella abortus* infection influenced the results of the milk tests in this investigation, however, because all the animals in both herds had been repeatedly negative to blood-tests.

As to the lactation period and the stage of lactation of the cows in this group, one was in the first week of the fifth lactation period, and four were in the 44th to the 49th week of lactation, two of these being in the second lactation period, one in the fifth and one in the seventh. One of the negative cows was in the second week of the fourth lactation period.

The milk production of the cows in this group was, in general, quite as satisfactory as that of the non-infected cows.

With regard to the previous history of the cows included in this investigation as to mastitis, we have no definite information. No records were kept of the occurrence of this disease in either of the herds to which the cows belonged but the herdsman in charge of the herd to which group B belonged, speaking from

memory, said he thought the two cows examined postmortem (P24 and 815) and six others (four of the infected and two of the non-infected) had had an attack of mastitis before the investigation began, but he could not remember which quarters were affected. The manager of the other herd was of the opinion that all cows in his herd that had had the disease had been disposed of before the beginning of the investigation.

No streptococci were found in the udders of 20 of the 46 cows included in the investigation, ten in group A and ten in group B. Six to nine samples of milk were drawn from each quarter and plated on blood-agar at intervals of four weeks and no colonies of streptococci appeared on any of the plates at any time. It would therefore appear safe to conclude that these cows were not infected with streptococci.

The cell-count, pH, and chlorine content of the milk samples were determined at the same time. A cell-count of a million or over was found in the milk from seven quarters in four cows, a pH above 6.8 in the milk from 35 quarters in 13 cows, and 0.14 or more chlorine in the milk from 40 quarters in 15 cows (table VI). The leukocytes were high in five samples from one quarter and in one sample from each of six quarters, the latter being in cows which at the time the sample was taken were in the later stages of lactation (36th to 50th week). As other data which will be discussed later indicate that the cell count increases with age, beginning with the fourth lactation period (table VII), it may be important to note here that only two of these cows were in the fourth lactation. All of the others were younger, 60 per cent being in the first lactation and 25 per cent in the second. Over 80 per cent of the quarters reacting to the pH and chlorine

TABLE VI—*Results of examination of non-infected cows.*

GROUP		LEUKOCYTES (1,000,000 OR OVER)		pH (ABOVE 6.8)		CHLORINE (0.14 OR OVER)		INDURATION		FLAKES	
		No.	%	No.	%	No.	%	No.	%	No.	%
A	Quarters	2	2.5	3	3.7	7	8.7	13	16.2	7	8.7
	Cows	1	5.0	3	15.0	5	25.0	8	40.0	5	25.0
B	Quarters	5	6.2	32	40.0	33	41.2	3	3.7	2	2.5
	Cows	3	15.0	10	50.0	10	50.0	3	15.0	2	10.0
A and B	Quarters	7	8.7	35	43.7	40	50.0	16	20.0	9	11.2
	Cows	4	20.0	13	65.0	15	75.0	11	55.0	7	35.0

tests were in cows in group B. No cause for this difference between the two groups was apparent but the number of positive reactions was unusual.

TABLE VII—*Comparison of the leukocyte count, pH and chlorine content with stage of lactation.*

SOURCE	STAGE OF LACTATION	MILK SAMPLES EXAMINED	LEUKOCYTES (1,000,000 OR OVER)		pH (ABOVE 6.8)		CHLORINE (0.14 OR OVER)	
			No.	%	No.	%	No.	%
From non-infected, non-indurated quarters	1st and 2nd weeks	14	2	14.2	0	0.0	0	0.0
	3rd to 39th week	692	53	7.6	36	67.9	34	64.0
	40th week and after	102	10	9.8	8	80.0	8	80.0
	Totals	808	65	8.0	44	67.6	42	64.3
From infected, non-indurated quarters	1st and 2nd weeks	3	1	33.3	0	0.0	0	0.0
	3rd to 39th week	93	35	37.6	14	40.0	16	45.7
	40th week and after	20	7	35.0	6	85.7	7	100.0
	Totals	116	43	37.0	20	46.5	23	53.4

Small flakes were observed in the milk of five cows in group A and two cows in group B; from two quarters in each of two of these cows and from one quarter in each of the other five. They were of the same appearance as those seen in the milk of the infected cows and were present only in the first few streams of milk. Eight of the cows in group A had small areas of induration in 13 quarters like those found in the infected cows and three cows in group B had three quarters with similar areas (table VI). There was no evidence of any correlation between the incidence of induration and flakes on the one hand and the milk changes on the other.

It will be noticed that the same changes were found in the milk and udders of the non-infected cows as in the infected, but that the incidence was somewhat lower. Seelemann[8] has noted an increase in cells, pH, and similar changes in the milk when streptococci were not present. In some cows the changes persisted for a long time and in others they were only temporary. In the latter case they may have been due to estrum, irregularities in

milking, injuries from pressure, chilling and other influences. According to Steck,[4-6] such changes in the milk may be due to the organisms which are ordinarily present in the normal udder. The activities of these organisms have a mild stimulant or irritant action on the udder, which causes changes in the secretion of greater or less degree, depending on the number and the species predominating. Steck classes the mastitis streptococci with the bacteria which may be present in the normal udder but states that their action on the udder is more pronounced than that of the others, the leukocyte content of the milk and the other changes being more marked.

A comparison of the results of the three tests used in this investigation on the milk samples from the quarters infected with streptococci with those of the milk samples from non-infected quarters shows that a much higher percentage of the samples from the infected quarters reacted positively. Leaving out of consideration the samples from the quarters in which induration was detected, 37 per cent of the samples from the infected quarters contained a million or more cells, as compared with 8 per cent of the samples from the non-infected quarters. Forty-six and five-tenths per cent of the infected samples had a pH above 6.8 and 53.4 per cent had 0.14 or more chlorine. The proportion of the non-infected samples which had a high pH and a high chlorine content was greater, 67.6 and 64.3 per cent respectively, due principally to the reaction of the samples from the cows in group B (table VII).

Comparing the test results with the lactation period, it is found that the percentage of positive reactions in the samples from the non-infected, non-indurated quarters shows a marked increase beginning with the fourth lactation, which is in agreement with the observation of Russell and Hoffmann.[18] A similar trend is also shown by the samples from the infected quarters but it is not so regular.

A number of investigators (Rühm, Pusch, Hewitt, Villar and Revis, and Gminder, cited by Steck[4-6] and Seelemann[8]) have observed an increase in leukocytes, pH, chlorine and other constituents of milk in the early and late stages of lactation while others (Russell and Hoffmann,[18] and Savage[19]) did not note any definite relation. In our examination of milk samples from non-infected, non-indurated quarters, 14.2 per cent of the samples from cows in the first two weeks of lactation had a leukocyte count of a million or over, 9.8 per cent of those from cows in the 40th week of lactation or after, and 7.6 per cent of those from cows in the middle stages of lactation. None of the samples from the cows in

the early stages of lactation had a high pH or a high chlorine content, while 80 per cent of those in the later stages were positive to both tests; 67.9 per cent of the samples from the cows in the middle stages had a high pH and 64 per cent were high in chlorine. The percentage of samples from infected, non-indurated quarters with a leukocyte count of a million or over was nearly the same for the different stages of lactation. A greater percentage of samples from cows in the 40th week of lactation or later had a higher pH and chlorine content than those from cows in the middle stages of lactation, but the number of samples examined was so small that the results cannot be considered indicative (table VII). The results of the tests on the non-infected samples would seem to indicate, however, that it would be advisable to consider the period of lactation and the stage of lactation in interpreting these tests.

The presence of a limited area of induration in the udder appears to have less effect on the leukocyte count and the pH than infection with streptococci. Only 6 per cent of the samples from the non-indurated, non-infected quarters and 9.7 per cent of those from the indurated, non-infected quarters had a cell-count of over a million, as compared with 49.1 per cent of the samples from the non-indurated, infected quarters and 58 per cent of those from the indurated, infected quarters. The effect on the pH was similar, the latter being above 6.8 in 14.5 per cent of the samples from the non-indurated, non-infected quarters, in 18 per cent of those from the indurated, non-infected quarters, 45.8 per cent of the samples from the non-indurated, infected quarters and 47 per cent of those from the indurated, infected quarters. The results of the chlorine test, however, were somewhat different, a higher percentage of samples from the non-indurated, infected quarters having a chlorine content of 0.14 or over than of those from the indurated, infected quarters: 65.8 as against 54.4. The results of the chlorine test on the samples from the non-infected quarters, however, were similar to those of the other tests, a higher percentage of samples from the indurated quarters being positive than from the non-indurated quarters: 26.7 of the former and 20.1 of the latter (table VIII). The fact that there are more cows in the later lactation periods (older cows) in the infected group need not be considered in connection with these results because the samples from non-infected quarters include those from the 29 non-infected quarters in the infected cows as well as those from the cows with all quarters free of streptococci.

TABLE VIII—*Comparison of the leukocyte count, pH and chlorine content with condition of the udder.*

SOURCE OF MILK SAMPLES	CONDITION OF UDDER QUARTERS	SAMPLES EXAMINED	LEUKOCYTES (1,000,000 OR OVER)		pH (ABOVE 6.8)		CHLORINE (0.14 OR OVER)	
			No.	%	No.	%	No.	%
From non-infected quarters..	No induration...	834	50	6.0	121	14.5	168	20.1
	Indurated areas.	299	29	9.7	54	18.0	80	26.7
From infected quarters..	No induration...	120	59	49.1	55	45.8	79	65.8
	Indurated areas.	136	79	58.0	64	47.0	74	54.4

SUMMARY OF RESULTS

This investigation has probably not been sufficiently extensive to justify the use of the results as a basis for final conclusions, but the information obtained is highly suggestive and may be summarized as follows:

1. Streptococci similar to the mastitis type were present in the udders of a number of cows for a considerable period without the development of clinical mastitis.

2. The leukocyte count was a million or over, the pH above 6.8, or the chlorine content 0.14 or over, and mastitis streptococci were present, in the milk of cows which did not develop clinical mastitis.

3. These changes frequently did not occur in the milk when streptococci were present, and yet did occur when streptococci were not present.

4. The leukocyte count, pH, and chlorine content of the milk of cows with normal udders free of streptococci more often exceeded the limits stated above in the early and late stages of lactation than in the intermediate stages.

5. The same changes were observed more often in the milk of cows with non-infected normal udders which were in the later periods of lactation, from the fourth or fifth on (older cows), than in the earlier periods (younger cows).

6. These observations raise the question as to whether the examination of milk for streptococci, and to determine the leukocyte count, pH, or chlorine content, should be depended upon as the sole means of deciding if a cow is suitable to produce market milk although such examinations, in connection with other methods, may be helpful in diagnosis, and may also be of assistance in

controlling the spread of streptococcic infection, thus preventing the development of clinical mastitis.

7. From our study, it would appear that the period of lactation and the stage of lactation should be considered in interpreting the results of the laboratory tests of milk

8. Limited areas of induration were found in udder quarters which were not infected with streptococci as well as in those which were infected with these organisms, but much less frequently in the former than in the latter.

9. Small flakes were observed infrequently in the first streams of milk from non-infected as well as infected quarters but they appeared in the milk of more of the infected quarters than the non-infected.

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Appropriate License Plate

Whether accidental or intentional, the Creely Dog Hospital, San Francisco, Calif., has acquired a particularly appropriate automobile license plate: 4 K 9. So unusual is it that Ripley has mentioned it in his "Believe It or Not" series.

THE PLACE OF THE WHOLE-BLOOD TEST IN A PULLORUM DISEASE-ERADICATION PROGRAM*

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The whole-blood agglutination test for pullorum disease, first described by Bunyea, Hall and Dorset,[1] in 1929, failed to give any assurance of being of much practical value until 1931, when these investigators, in collaboration with Schaffer and MacDonald,[2] introduced the stained antigen and took the steps necessary to assure its production as a standardized biological product. The whole-blood test with stained antigen has unquestionably met with the popular approval of the industry and is being applied on a large scale by poultrymen. The major portion of this testing has been done as a conscientious endeavor to eradicate the disease from breeding flocks as economically as possible. There are those, however, who have followed and still are following the rather short-sighted policy of conducting the test solely for advertising purposes. Regardless of the motives underlying a pullorum disease-testing program, it is becoming increasingly evident that live stock sanitary officials who have not already done so will be called upon to take cognizance of the whole-blood agglutination test method as a pullorum disease control procedure.

A decision concerning the place of the whole-blood agglutination test in a pullorum disease-eradication program must be guided by a very definite knowledge as to the probable presence or absence of pullorum-infected birds in tested flocks. Is the efficiency of the whole-blood test such that it will detect all pullorum-infected birds of a given flock? What factors influence the efficiency of the test as conducted under field conditions and may technical skill be improved to the point where comparative test results will be uniform? These very pertinent questions, either direct or implied, were to serve as the basis for the experimental studies reported herein of the efficiency of the whole-blood agglutination test as conducted on five poultry farms. The owners of these farms, after having experienced rather heavy losses from pullorum infection in baby chicks for several years, were desirous of eradicating it from their breeding flocks. Because of the cost of conducting the tube agglutination test, these men favored a trial of the whole-blood tests and agreed to coöperate to the fullest ex-

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tent in carrying out a program designed to show the efficiency of the test in the eradication of the disease.

The Testing Program

Beginning in September, 1932, group 1, comprising all fowls (male and female) which were to provide hatching eggs for the 1932-33 season, were tested by means of the whole-blood agglutination method. Testing of group 2 birds, comprising all yearling fowls (1932 pullets), was initiated during February or March of 1933; while testing of group 3, the progeny of group 1, was begun during May or June, 1933, when these birds were about four months old. After September, 1933, no untested fowls, except those too young to be tested, were permitted to be kept on the farm. The tests of any group were conducted at intervals of approximately one month, until one, and preferably two, completely negative tests were obtained. All fowls which gave either a positive or suspicious reaction at each test were immediately removed from the flock and premises.

After a group had passed one completely negative whole-blood test, it was viewed as having qualified for the experimental study to determine its status relative to the presence of pullorum-infected birds. To determine this, when the next regular whole-blood test of the group was conducted, blood samples were also obtained from each bird and forwarded to the laboratory for the purpose of check-testing by means of the tube agglutination method. All positive or suspicious reactors to either one or both tests were obtained for further serological studies and bacteriological examination. If pullorum infection was demonstrated in any of the birds thus removed, the group was usually retested by the whole-blood method alone until it had qualified for another comparative test. Occasionally combination tests were conducted before a group had qualified by a negative whole-blood test. This was particularly true in the various group 3 series of birds at a time when we wished to collect more data on comparative tests than our plans as originally outlined would permit.

After group 1 birds had been tested over a period of one year, they were permitted to cohabit with group 2. While the ideal arrangement would have kept these groups as separate units throughout the experiment, the advantage of so doing would not justify the expense which such an arrangement would have necessitated.

The flock-owners coöperated splendidly in such matters as the purchase of hatching eggs or replacements with baby chicks or adult stock, the disinfection of incubator and brooding equipment, and the refusal to do custom-hatching.

The conducting of the whole-blood test, removal of reactors and the fulfillment of the requirements of the pullorum disease-eradication program on each farm during the two-year period of this study were under the direct supervision of Mr. E. R. Temperli, whose untiring efforts and strict attention to detail in procuring the field data served as a most valuable contribution to this study.

WHOLE-BLOOD ANTIGEN AND FIELD-TEST TECHNIC

The antigen used for the whole-blood test was prepared in our laboratory according to the rules as set forth by the Chief of the Bureau of Animal Industry, under date of September 28, 1931 (U. S. Patent 1,816,026). During the course of the experiment and more particularly during our laboratory studies, the test results with this antigen (California-prepared) compared most favorably with those obtained from federal Bureau of Animal Industry antigen secured through the courtesy of Doctor Dorset.

In order that the whole-blood test results would be comparable to those obtained by the inexperienced poultryman, neither Mr. Temperli nor the flock-owners were given any special instruction at the start on the technical phases of the test. We were interested further in determining whether the test efficiency would increase as those conducting it became more experienced.

The whole-blood test throughout this experiment was made by the addition of one drop of blood to one drop of stained antigen and stirred on a stationary plate until a homogenous mixture was obtained. In previously conducted studies of their comparative accuracy, more accurate results were secured with a stationary plate than when it was rocked or rotated. After analyzing the data obtained from the first series of comparative tests and bacteriological examinations, certain improvements were made in the technic and equipment used. It was determined that negative readings of the test could not be considered as final until after at least six, and preferably eight, minutes of incubation; in fact, our data show a small number of actively infected birds in which no evidence of a whole-blood agglutination appeared until after ten or twelve minutes of incubation. Holding the test mixture under observation for this length of time necessitated adequate protection against evaporation by maintaining incubation under glass plates. An electrically-lighted testing-box was constructed, by which a constant temperature of 80° F. could be maintained and uniform light for reading tests was provided.

When the whole-blood test alone was applied, all birds which gave any indication of a reaction were designated as positive and

removed from the flock and marketed. When the combination (whole-blood and tube agglutination) tests were applied, the degree of the whole-blood test reaction was designated in terms of positive or suspicious. This was done for the purpose of a more detailed comparison of results of retests of the birds by the whole-blood and tube methods which were conducted prior to autopsy.

At the time of combination tests, birds which reacted to the tube agglutination method were removed on the basis of their reaction in a 1:25 dilution. Sera which gave a complete agglutination were designated as positive; all partial agglutinations were regarded as suspicious. These interpretations, particularly of what constituted a suspicious reaction, should be kept in mind, since this so-called "close reading" will be referred to in our discussion of the comparative test results. The interpretation of the tube agglutination retests conducted just prior to autopsy of the bird was based upon the titre of serum-antigen dilutions of 1:25, 1:50, 1:100 and 1:200.

The degree of agglutination in each dilution was designated as follows: complete, 4+; incomplete, 3+; partial, 2+; slight agglutination, 1+. In this, as in the routine test, a complete agglutination in the 1:25 dilution was interpreted as a positive reaction. While the probability of demonstrating active infection increased with the agglutinin titre in higher dilutions, *Salmonella pullorum* was not infrequently isolated from birds whose serum gave a complete agglutination in a dilution of 1:25 and a slight (1+) or negative reaction at 1:50. In the 1:25 dilution an incomplete agglutination (3+) was interpreted as highly suspicious, a partial agglutination (2+) as slightly suspicious, and a slight agglutination (1+) as non-specific. Final readings of the tube agglutination tests were made after an incubation period of 24 hours at 37.5° C.

In justice to either test, no attempt was made to evaluate their efficiency solely on the basis of direct comparison. Such a standard, frequently used in the past, is erroneous and would have been particularly so in this experiment where the specific function of the routine tube test was that of removing all birds which gave any indication of agglutinins. It was recognized that on this basis probably many of the birds which were classified as suspicious to the routine tube test would fail to react on retest in the laboratory, but at the same time would serve as the only practical method of removing all actively infected birds and thus afford a basis for determining the efficiency of the whole-blood test method.

Bacteriological examination of the birds which were designated as positive or suspicious reactors to either one or both of the combination tests (whole-blood and tube agglutination) was usually completed within a period of seven days after the test. Cultures on fresh blood-agar plates were made from the lungs, pericardial fluid, heart-blood, liver, spleen and ovary. A diligent search for detached, inspissated ova and abdominal cysts was rewarded in many instances by the isolation of *S. pullorum* in an otherwise negative autopsy. Isolations were made from some spleens which failed to show any gross pathological lesions.

THE EXPERIMENT DATA

As indicated in the plan of the experiment, the data presented in tables I to V comprise the record of the whole-blood test results in 14 groups of birds on five farms and represents a total of 91,779 test birds, of which 42,214 were also tested by means of the routine tube test method. While the decrease in the percentage of reactors detected during the frequent retests may indicate a relatively high efficiency for the whole-blood test method, such an interpretation becomes questionable when the test results are compared with the tube test results and bacteriological findings in birds autopsied.

The degree of efficiency of the various test methods is most critically analyzed by a comparison of the test results and autopsy findings on the 405 birds removed as reactors. The serological retest results obtained in the laboratory just prior to autopsy of the birds are of particular interest because they represent the difference between the efficiency of the test methods as conducted under field and laboratory conditions. The laboratory retest results should represent each test at its highest technical efficiency and should serve as the basis for a direct comparison of the relative efficiency of the whole-blood and the standard tube agglutination test methods.

Before taking up a brief discussion of the detailed data as presented in tables I to V, a few explanatory remarks concerning the method of presentation may be in order. For example, referring to table I, 564 birds comprising group 1 of flock A, were tested by both the whole-blood and routine tube test methods on December 1, 1932, having so qualified by virtue of one negative whole-blood test under date of November 10, 1932. On December 1, the whole-blood test failed again to detect any reactors, but the routine tube test designated seven birds as positive and six as suspicious. On retest in the laboratory prior to autopsy, five of the seven positive birds were designated as positive to the

TABLE I—*Data on flock A.*

Test Number	Date	Group	Birds Tested	Reactors to Routine Tests					Laboratory Retests of Reactors to Routine Tests						Salmonella Pullorum	
				Whole Blood			Tube		Whole Blood			Tube			Isolated	Not Isolated
				Pos.	Susp.	% Pos.	Pos.	Susp.	Pos.	Susp.	Neg.	Pos.	Susp.	Neg.		
1	Sept. 27/32	1[1]	633	42	0	6.6										
2	Oct. 25/32	1	583	3	0	0.5										
3	Nov. 10/32	1	570	0	0											
4	Dec. 1/32	1	564	0	0		7		5			5			4	1
										1		1			1	
											1	1			1	
								6[4]			5	1			1	
													4		1	3
5	June 10/33	1	404	2	0	0.5										
6	July 8/33	1	318	0	0											
7	Sept. 13/33	1	232	0	1		0	0			1			1		1
				0	0			2	1				1		1	
										1			1			1
8	Nov. 19/33	1	211	0	0		0	0								
1	March 4/33	2[2]	1461	80	0	5.4										
2	April 8/33	2	1325	49	0	3.7										
3	May 6/33	2	1104	13	0	1.1										
4	June 10/33	2	976	9	0	0.9										
5	July 8/33	2	915	0	0											
6	Sept. 13/33	2	843	0	2		0	0			2			2		2
				0	0		10		6			5			2	3
													1			1
										3		2	1		3	
											1	1			1	
								9[4]	2					2		2
											6		3		2	1
														3		3
7	Nov. 19/33	2	795	0	0		0	0								
1	Jan. 28/34	3[3]	1610	0	0		0	0								
	Sept. 10/34	1-2-3	1300	0	0		0	0								

[1]Fowls of this group hatched in spring of 1931.
[2]Fowls of this group hatched in spring of 1932.
[3]Fowls of this group hatched in spring of 1933 and are progeny of group 1. (Hatching eggs collected after Jan. 5, 1932.)
[4]One bird died on premises. Not retested or examined.

TABLE II—*Data on flock B.*

Test Number	Date	Group	Birds Tested	Reactors to Routine Tests					Laboratory Retests of Reactors to Routine Tests						Salmonella Pullorum	
				Whole Blood			Tube Test		Whole Blood			Tube Test			Isolated	Not Isolated
				Pos.	Susp.	%Pos.	Pos.	Susp.	Pos.	Susp.	Neg.	Pos.	Susp.	Neg.		
1	Sept. 13/32	1[1]	897	21	0	2.3										
2	Oct. 10/32	1	869	3	0	0.3										
3	Oct. 31/32	1	856	0	0	0										
4	Nov. 22/32	1	849					14					14		1	13
5	Sept. 11/33	1	391	0	0	0										
6	Oct. 10/33	1	390	0	0	0		4					1	3		4
1	Jan. 29/33	2[2]	2119	0	5	0.2										
2	Sept. 11/33	2	750	0	2[4]	0.3				1		1			1	
3	Oct. 10/33	2	761	0	6	0.8	4		4			4			4	
											2			2		2
				0	0	0	5		5			5			5	
				0	0	0		4		1	3		1	3		4
4	Nov. 28/33	2	706	0	0	0	0	0								
7-5	Sept. 16/34	1-2	887	0	0	0	0	0								
1	Feb. 12/34	3[3]	1730	0	0	0	2			2			1	1		2
2	Sept. 16/34	3	515	0	0	0	0	0								

[1]Fowls of this group hatched in spring of 1931.
[2]Fowls of this group hatched in spring of 1932.
[3]Progeny of group 1. (Hatched from eggs collected after Nov. 22, 1932.)
[4]One bird died on premises. Not retested or examined.

TABLE III—*Data on flock C.*

Test Number	Date	Group	Birds Tested	Reactors to Routine Tests					Laboratory Retests of Reactors to Routine Tests						Salmonella Pullorum	
				Whole Blood			Tube Test		Whole Blood			Tube Test			Isolated	Not Isolated
				Pos.	Susp.	%Pos.	Pos.	Susp	Pos.	Susp.	Neg.	Pos.	Susp.	Neg.		
1	Sept. 28/32	1[1]	782	47	0	6.0										
2	Oct. 26/32	1	718	9	0	1.3										
3	Nov. 10/32	1	698	0	0	0										
4	Dec. 7/32	1	698	0	0	0	9		1			1			1	
										1			1		1	
											7	5	1		6	
														1		1
				0	0			12			12	3			1	2
													7			7
														2		2
5	Sept. 26/33	1	417	0	2		0	0			2			2		2
6	Oct. 25/33	1	470	0	0	0	0	0								
7	May 20/34	1	292	0	0	0	0	0								
1	Nov. 8/33	3A[2]	387	2	0				2			2			2	
2	Dec. 12/33	3A	371	2			2		1	1		2			2	
					2			2	1			1			1	
											1			1		1
				0	0	0	2		2			2			2	
1	Apr. 11/34	3B[3]	463	0	0	0	1	0		1			1			1
	May 20/34	3A+B	714	0	0	0	1	1		2			2			2
	Sept. 19/34	3A+B	753	0	0	0	0	0								

[1]Fowls of this group hatched in spring of 1931.
[2]Fowls of this group (3A) hatched in December of 1932, from group 1 eggs collected after Nov. 10, 1932.
[3]Fowls of this group (3B) hatched in April of 1933, from group 1 eggs collected after Dec. 7, 1932.

TABLE IV—*Data on flock D.*

Test Number	Date	Group	Birds Tested	Reactors to Routine Tests					Laboratory Retests of Reactors to Routine Tests						Salmonella Pullorum	
				Whole Blood			Tube Test		Whole Blood			Tube Test			Isolated	Not Isolated
				Pos	Susp	%Pos	Pos	Susp	Pos	Susp	Neg	Pos	Susp	Neg		
1	Sept 19/32	1[1]	4169	301	0	7.3										
2	Oct 18/32	1	3735	57	0	1.5										
3	Nov 8/32	1	3471	16		0.5										
4	Nov 29/32	1	3239	6		0.2										
5	Dec 14/32	1	1909	9		0.5										
6	Sept 14/33	1	1461	20	0	1.4	14		14		6	14		6	14	6
7	Oct 10/33	1	1262	1	0				1			1			1	
8	Nov 7/33	1	1217	8	0		5		4			4		1	4	1
										1	3	1		2	1	2
				0	0		7			1		1			1	
											6	3	3		2	1 3
				0	0			5		1		1				1
											4		1	3		1 3
1	Mar 25/33	2[2]	1674	34	0	2.0										
2	May 7/33	2	1541	27	0	1.8										
3	June 17/33	2	1223	13	0	1.1										
4	July 17/33	2	1049	2	0	0.2										
5	Sept 14/33	2	557	0	0	0										
6	Oct 17/33	2	495	2	0	0.4	2		1			1			1	
										1		1			1	
				0	0		8		3			3			3	
											5		1		1	
												4			2	2
				0	0			4			4		2			2 2
7	Dec 3/33	2	411	4		1.0					4			4		4
				0	0		1				1	1			1	
8	Mar 25/34	2	257	0	0		0	0								
9	April 22/34	1-2	921	0	0		0	0								
10	Sept 23/34	1-2	577	0	0		0	0								
1	Feb 25/34	3[3]	2534	36	0	1.4	17		16			16			14	2
										2		1		1	1	1
											18			18	1	17
				0	0		16		9			9			9	
										5		4	1		5	
											2	1	1		2	
				0	0			2			2	1		1	1	1
2	Mar 25/34	3	2184	8	0	0.4	8		8			8			8	
				0	0		4		1			1				1
										1		1			1	
											2	1	1		1	1
3	May 6/34	3	2027	2	0	0.1	1		1			1			1	
											1			1		1
4	Sept 26/34	3	1303	0	0		0	0								

[1]Fowls of group 1 were hatched in spring of 1931.
[2]Fowls of group 2 were hatched in spring of 1932.
[3]Fowls of group 3 are the progeny of group 1 from eggs collected after Dec. 14, 1932.

TABLE V—*Data on flock E.*

Test Number	Date	Group	Birds Tested	Reactors to Routine Tests: Whole Blood Pos	Whole Blood Susp	Whole Blood % Pos	Tube Test Pos	Tube Test Susp	Laboratory Retests of Reactors to Routine Tests: Whole Blood Pos	Whole Blood Susp	Whole Blood Neg	Tube Test Pos	Tube Test Susp	Tube Test Neg	Salmonella Pullorum: Isolated	Salmonella Pullorum: Not Isolated
1	Sept 14/32	1[1]	1331	149		11.2										
2	Oct 11/32	1	1139	58		5.1										
3	Nov 1/32	1	1046	10		0.9										
4	Nov 22/32	1	1003	0	0	0										
5	Dec 5/32	1	966	0	0	0	6[5]		2			2			2	
											3	3			2	1
				0	0			22[6]	3			1			1	
													2			2
										3					1	2
											14	3			2	1
													5		1	4
														6	1	5
6	Jan 20/33	1	905	11	0	1.2			4		7		4	7	2	9
7	July 19/33	1	594	13	0	2.6										
1	Feb 15/33	2[2]	1459	74		5.1										
2	Apr 11/33	2	1169	74		6.3										
3	May 20/33	2	1013	55		5.4										
4	June 24/33	2	862	19		2.2										
5	July 19/33	2	793	4		0.5										
8-6	Sept 30/33	1-2	1047	10		0.9	7		6			6			6	
										1		1			1	
											3			3		3
9-7	Oct 21/33	1-2	1040	3		0.3	1		1			1			1	
											2			2		2
10-8	Nov 21/33	1-2	1015	1		0.1	0	0			1			1		1
				0	0		19		6			6			6	
										6		6			5	1
											7	4			4	
													3		2	1
				0	0			12	1			1			1	
										2		1	1		1	1
											9	4		2	4	2
													3		1	2
11-9	Apr 18/34	1-2	754	2		0.3	1		1			1			1	
											1			1		1
				0	0		12		5			4			3	1
										2			1		1	1
											5	1			1	1
													3	1	1	2
				0	0			4		2			2			2
											2			2		2
12-10	May 23/34	1-2	599	0	0		0	0								
13-11	June 20/34	1-2	563	0	0		0	0								
14-12	Oct 3/34	1-2	531	0	0		0	0								
1	Sept 30/33	3A[3]	542	13		2.4										
2	Oct 21/33	3A	536	0	0											
3	Dec 5/33	3A	528	5		0.9	5		5			5			5	
					3			3		1			1			1
											2			2		2
				0	0		8		6			6			6	
										1		1				1
													1			1
				0	0			7[7]			6	2			1	1
													1		1	
														3		3
4-1	Apr 5/34	3A-3B[4]	1065	14		1.3	7		7			7			7	
											7			7		7
				0	0		24[7]		14			12			11	1
													2		2	
										3		2		1	2	1
											3		2		2	
														1		1
				0	0			11	5			4			3	1
													1			1
										1			1			1
											5			3	2	1
																2
5-2	May 23/34	3A-3B	789	1		0.1	1		1			1			1	
				0	0		4		3			3			3	
											1			1		1
				0	0			1			1	1			1	
6-3	June 20/34	3A-3B	654	0	0		0	0								
7-4	Oct 3/34	3A-3B	401	0	0		0	0								

[1]Fowls of group 1 were hatched in spring of 1931.
[2]Fowls of group 2 were hatched in spring of 1932.
[3]Fowls of group 3A are the progeny of group 1 from eggs collected after Dec. 5, 1932.
[4]Fowls of group 3B are the progeny of group 1 from eggs collected after Jan. 20, 1933.
[5]One bird died on the premises. Not retested or examined.
[6]Two birds died on the premises. Not retested or examined.
[7]Four birds died on the premises. Not retested or examined.

whole-blood test, while one was suspicious and the remaining bird was negative.

Each of the five birds which were positive to the whole-blood test was also positive to the tube agglutination test, and in four of the number active *S. pullorum* infection was demonstrated. The birds which gave a suspicious and a negative reaction, respectively, to the whole-blood test were positive to the tube test and were actively infected birds. One of the six birds which gave a suspicious reaction to the routine tube test died and was not included in the shipment to the laboratory. Each of the five birds which were forwarded gave a negative reaction to the laboratory whole-blood test. The tube retest designated one non-infected bird as positive, while infection was demonstrated in one of the remaining four birds classified as suspicious reactors.

After another negative whole-blood test on July 8, 1933, group 1 received a combination test on September 13, 1933. One bird designated as suspicious to the whole-blood test was negative to the routine tube test, to laboratory retests by both the whole-blood and tube methods and *S. pullorum* infection was not demonstrated at autopsy. Retest of the two birds designated as suspicious to the routine tube test showed one to be positive to the whole-blood method, suspicious to the standard tube test and pullorum-infected. The second bird was suspicious to both retests and non-infected.

This demonstration of reactors and *S. pullorum*-infected birds in a group which had passed two negative whole-blood tests on two different occasions is typical of the observations in eight other groups of this experiment and in certain other flocks in which we have taken the occasion to determine the efficiency of the whole-blood test as conducted under field conditions by the experienced operator. The whole-blood test and routine tube test results were in agreement only in case no birds gave a positive reaction to either test or where the birds tested represented the progeny of negative groups. These observations indicate the limitation of the whole-blood test in a program the objective of which is pullorum disease eradication.

A comparison of the efficiency of the whole-blood test as conducted under field and laboratory conditions is presented in table VI. In this summary, the test efficiency is based upon the serological results obtained on the 217 *S. pullorum*-infected birds and to a lesser extent upon the apparently unwarranted removal of 188 non-infected birds. It will be observed that the routine whole-blood test detected only 84 (75 positive and 9 suspicious reactions) out of the 217 infected birds, and 133 infected birds gave

negative reactions, a test efficiency of 38.71 per cent. The whole-blood test as conducted in the laboratory detected 166 of the 217 birds by 140 positive and 26 suspicious reactions, with the remaining 51 birds undetected, a test efficiency of 76.5 per cent.

The routine tube test detected 216 and the standard tube method 215 of the 217 infected birds, representing a test efficiency of 99.53 and 99.07 per cent, respectively. The tube test method, both routine and laboratory retest, failed to detect one infected bird which had given strongly positive reactions to the whole-blood test. The second infected bird which failed to react to the laboratory tube and whole-blood retests had been removed from the flock on the basis of a partial reaction to the routine tube test and is probably representative of the receding phase of an undulating agglutination titre.

TABLE VI—*Correlation between the serological test reactions and the isolation of S. pullorum in the 405 birds removed as reactors to either the whole-blood or the tube test, alone or in combination.*

S. Pullorum Isolated (Fowls)		Whole-Blood Test						Tube Test					
		Routine			Laboratory Retest			Routine			Laboratory Retest		
+	−	Pos.	Susp.	Neg.	Pos.	Susp.	Neg.	Pos.	Susp.	Neg.	Pos.	Susp.	Neg.
217		75	9	133	140	26	51	188	28	1	190	25	2
	188	57	11	120	21	19	148	37	90	61	25	62	101

In a final summation of the test efficiency, one cannot disregard the unwarranted removal of non-infected birds. Considering the economic value of the individual, errors in this respect are within certain limits relatively insignificant when compared to the potential hazards of failure to detect infected birds. The relatively large number of 188 non-infected, and to a large extent non-reacting, birds on retests by the tube method in a total of 405 reactors to one or both routine tests is due to some extent to misinterpretation of atypical reactions to the whole-blood test, but more particularly to the intentionally liberal interpretation of what constituted a suspicious reaction to the routine tube test. The fact that subsequent retests usually failed to disclose reacting birds among those previously designated as suspicious would seem to justify the interpretations referred to.

Discussion

Before attempting a practical interpretation of this or other experiment data, we must recognize that the whole-blood test has

been favorably received by the poultry industry and will continue to be used by it in either a conscientious endeavor in eradicating or controlling pullorum disease or for purposes of advertising under the now rather meaningless term of "blood-tested." Regardless of the motive which prompts its use, it is becoming increasingly evident that live stock sanitary officials will be called upon to decide if and under what conditions the whole-blood test results will receive official recognition. While one may certify that the flock-owner has complied with the requirements of a given pullorum-testing program, worth-while official certification of this should be reserved for those flocks in which the disease has been completely eradicated.

Inefficiency of the serological test itself or its method of application which results in failure to detect all pullorum-infected adult birds within a breeding flock is apt to result in the continued dissemination of the infection and destroy confidence in the results which may otherwise be obtained. The discriminating purchaser of hatching eggs or baby chicks is less interested in the term "blood-tested," but in pullorum disease-free progeny, and is beginning to inquire relative to the testing method, by whom and under whose supervision it was applied.

A review of the data of this experiment reveals these pertinent facts:

1. Repeated testing by the whole-blood method, although rendering the flocks completely negative to that test, failed in each instance to detect all infected fowls or all that would react to the tube test.

2. The whole-blood test was more efficient when conducted in the laboratory than when conducted in the field.

3. Repeated testing by the whole-blood method reduced the number of infected birds in the various flocks to 1 per cent or less.

4. In several instances after a single test by the tube method the flocks which were negative to the whole-blood test continued negative to both tests.

5. Progeny of the groups of fowls that became negative to tests by both methods were pullorum disease-free.

Although the primary purpose of this experiment was to contribute data relative to the efficiency of the whole-blood test, such a study necessarily included observations of those major factors which may influence the test results. Since the whole-blood test is designed for field use, the explanation of the differences in the results obtained with it in the field and laboratory is of particular importance. The greater efficiency of the whole-blood method

conducted in the laboratory cannot be attributed to differences in antigen or equipment because these were identical in each instance. Inexperience of those conducting the field tests is ruled out by the fact that when laboratory retests were conducted, several thousand field tests had been made and cognizance had been taken of the importance of such factors as holding the tests for at least six to eight minutes before final reading, uniformity of light for reading, maintenance of temperature at about 80° F., and other points of a technical nature already referred to.

Differences in interpretation of the tests by those inexperienced in making them undoubtedly account for some of the difference between field and laboratory results, but the writers have reason to believe that this factor was of minor importance in this case. The main causes for the lower efficiency of the field tests would, therefore, appear to be factors peculiar to the field conditions. The first among these to be mentioned is that the number tested at a time was, in all cases, approximately 1,000 or a greater number. A natural consequence of this was a tendency to make speed which, it seems reasonable to assume, was attained at the sacrifice of accuracy. This is in sharp contrast to the laboratory observations, which were made very deliberately and on but a few fowls at a time.

Another factor of possible import is uncontrollable environmental conditions, particularly those of atmospheric humidity and temperatures. The relative humidity in the field was usually low but the temperatures varied, with the season and time of day, from around freezing to in excess of 100° F. and, in many instances, varied between wide limits during the time a single group of birds was being tested. At the laboratory, the relative humidity was always high and the temperature constant between 70 and 80° F. The influences of the above mentioned factors on field test results cannot be exactly evaluated but, in the opinion of the writers, they are real and will be operative wherever the whole-blood test is conducted. It seems highly important, therefore, that whole-blood tests be conducted or closely supervised by trained persons who are fully conscious of all factors that may influence the efficiency of the test and that laboratory facilities for check-testing be available and utilized.

Despite its apparent limitations we feel that the whole-blood test has a definite place in a pullorum disease-eradication program and are, therefore, encouraging its supervised use in California flocks. Our experiment data indicate that the test will serve as a valuable adjunct in preparing flocks for final accreditation on the basis of the tube test. It serves this purpose admirably because it is easily adapted to a program of frequent retests

which is the basis upon which effective control and ultimate eradication are greatly enhanced. In this respect it is distinctly advantageous to the tube test method, both from the standpoint of the poultryman and the laboratory technician.

In the testing program incident to qualifying his flock for an official tube test, the poultryman will attain a greater appreciation of the benefits to be derived therefrom and will, therefore, coöperate to the fullest extent in a program of eradication. On the other hand, the progress of pullorum disease eradication on the basis of the standard tube test, now criticized as slow, expensive and, therefore, of limited scope, would be aided by reserving such tests to flocks which have qualified for it with two successive negative, officially supervised whole-blood tests. Such supplementary use of the whole-blood test would relieve the official testing laboratories of their present burden of preliminary testing and of all testing in those flocks the owners of which were insincere in their efforts at pullorum disease control. The combination test results and subsequent examination of reactors which might be made incident to following such a program would contribute much additional data on the efficiency of the whole-blood test method.

The present efficiency of the standard tube test method is the result of years of experimental study of those factors influencing the correlation between the test results and actual infection. Rather than accept the present apparent inefficiency of the whole-blood test as representing its final limitation, experimental studies should be continued. In the meantime, poultrymen should be acquainted with all the facts concerning the advantages and limitations of testing their flocks by the whole-blood method.

CONCLUSIONS

1. The efficiency of the whole-blood (stained antigen) test will not permit of its exclusive use in a program of pullorum disease eradication.

2. By retesting with the whole-blood method at short intervals, the number of pullorum-infected birds in a flock may be quickly reduced to 1 per cent or less and the flock in this way be prepared for accreditation or certification as pullorum disease-free on the basis of the more highly efficient tube test.

3. Official recognition should be reserved to the tube test results until the whole-blood test has been improved to the point that its efficiency equals that of the tube test.

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THE ISOLATION OF THE ORGANISM OF AVIAN TUBERCULOSIS FROM CATTLE*

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The infectiveness of *Mycobacterium tuberculosis avium* for certain mammals has been reported sufficiently often to indicate that this organism is capable of inducing lesions in a considerable number of domesticated species. The etiologic significance of the organism of avian tuberculosis has been demonstrated in horses, by Nocard,[1] M'Faydean,[2] and Watson and Heath[3]; in swine, by Van Es and Martin[4]; and in sheep, by Griffith,[5] and Harshfield and Roderick.[6] The work of Feldman indicates that dogs are highly resistant to this form of tuberculous infection.[7]

That cattle also are susceptible to infection by the bacilli of avian tuberculosis is evident from the reports of many workers in different parts of the world. One of the earliest instances of avian tuberculosis infection in a bovine animal was the case of Johne and Frothingham (1895).[8] These workers isolated, from a natural infection in a cow, organisms that by tests for pathogenicity were considered to belong to the avian variety. The organisms were not pathogenic for guinea pigs, and the lesions in the cow, from which the bacteria were obtained, were not considered typical of those of the bovine form of the infective agent.

Plum[9] quoted the observations of Bang (1908), who isolated the avian form of the tubercle bacillus from a bronchial lymph-node of a cow affected with pulmonary tuberculosis. Plum, in discussing this case, considered the possibility that the tuberculous lesions of the lungs might have been the result of the bovine form of the organism, but since the lymph-node yielded bacteria, which were markedly pathogenic for rabbits but not for guinea pigs, the presence of the bovine form of the infective agent seemed unlikely. De Jong[10] described the isolation of a strain of avian tubercle bacilli from a case of congenital tuberculosis of a calf. While this strain proved pathogenic for one of eight guinea pigs that were inoculated, the fact that it was pathogenic for rabbits, chickens and doves, and was culturally dissimilar to the bovine form of the organism, seemed to justify de Jong's conclusion that the organism was avian in type.

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The occurrence of abortion in cattle, which was caused by the organism of avian tuberculosis, was investigated by Plum.[12] This author observed that about 2 per cent of the cows slaughtered in Denmark had lesions of tuberculosis in the uterus. Material from the fetal membranes of 162 such cows was injected into laboratory animals with the following results. In ten cases the animals died of sepsis and 145 of the remaining 152 cases showed conclusive evidence of mammalian tuberculosis. Six of the remaining seven cases yielded bacteria of avian tuberculosis and the character of the infection of one was uncertain. Plum mentions the finding of tuberculous lesions in a calf that had been born prematurely from one of the six cows from which avian tubercle bacilli had been obtained. The mother of this calf (cow 6) proved to be infected with both the bovine and avian forms of the tubercle bacillus, the avian variety of the organism having been isolated from the uterine discharges. In all, Plum isolated the organism of avian tuberculosis from nine bovine animals, six cows and three calves.*

In a more recent report (1931) Plum[13] reported the occurrence of infection caused by the avian form of *M. tuberculosis* in three different herds of cattle.

Van Es and Martin[14] examined the lesions of 164 bovine animals in which gross morphologic evidence suggestive of tuberculosis was limited to solitary, isolated foci. Negative results were obtained in 49 cases, while 115 lesions were proved by animal inoculation tests to be definitely tuberculous. By the use of rabbits and chickens, the type of the infective agent also was determined with the following results: mammalian tubercle bacilli, 100 cases; avian tubercle bacilli, eleven cases; both avian and mammalian tubercle bacilli, four cases. The majority of the animals from which the avian form of the tubercle bacillus was obtained were adult cattle and the organism was secured from mesenteric lymph-nodes in ten of the eleven instances and once from a mass in the omentum.

The occurrence of avian tuberculous infection in cattle was also reported by Van Heelsbergen[15] from Holland. This author was of the opinion that the avian type of the infective agent probably occurs more frequently in cattle than is generally supposed. According to Van Heelsbergen, the lesions occur most frequently in the lymph-nodes of the digestive tract, although the lungs and liver may also be affected. Abortion resulting

*It is of interest to note that an instance of abortion in a sow, associated with an infection caused by the organism of avian tuberculosis, was recently reported by Werdelin.[12] Tuberculous lesions were present in the uterus, lumbar lymph-nodes, mesenteric lymph-nodes, and lungs.

from infection of the uterus by the avian tubercle bacillus was considered by Van Heelsbergen as one of the symptoms of avian tuberculosis infection in cows.

Wolters and Dehmel,[16] in Germany, were successful in recovering 23 strains of tubercle bacilli of the avian type from the milk and tracheal mucus of cows. The organisms were identified as being of avian type by the inoculation of white mice, guinea pigs, rabbits and chickens. The cultural characteristics also were studied. The infective bacteria were obtained from the following: nine strains from 400 specimens of mixed milk from nine different farms; ten strains from 143 specimens of milk from individual cows, and four strains from 80 specimens of mucus from the respiratory tract.

The occurrence of avian tuberculosis in cattle in Great Britain was reported by Minett[17] who obtained the provocative organism from three calves and two adult bovines belonging to three separate herds. In four of the animals the organisms were isolated from the lymph-nodes of the alimentary tract and in one case the bacteria were obtained from the mucosa of the intestine which, while hyperplastic in places, was otherwise apparently normal in appearance. The strains obtained from the calves were highly virulent for rabbits and chickens, while the two strains obtained from the adult cattle were of low virulence for these animals.*

Original Observations

The material constituting the basis of this report was obtained during the past two years from a near-by dairy herd that had been tested with tuberculin during a period of eleven years. Very few animals have been added to the herd through purchases, the only additions from outside sources being two cows and two bulls. At the present time most of the herd is made up of animals born on the premises.

During the past eleven years, a total of 21 animals have been removed from the herd and slaughtered on account of a positive tuberculin reaction (table I). None of these animals, when examined at necropsy, revealed generalized or progressive lesions of tuberculosis. In a considerable number, no visible lesions could be found, and the lesions in the others were usually of a solitary localized character, such as subcutaneous tuberculoid lesions (so-called "skin lesions") or suggestive changes of uncertain etiology in a few lymph-nodes.

*For additional references pertaining to the occurrence of organisms of avian tuberculosis in cattle, see Zeller, H.: Die Tuberkulose des Geflügels. Ergeb. d. allege. Path., xxvi (1932), pp. 804-876.

Material for this study was secured at the time of slaughter from eleven of the animals, which had reacted to tuberculin during the past two years, in an attempt to demonstrate the presence of *M. tuberculosis.*

TABLE I—*Number of cattle in the herd during eleven-year period and the number of reactors to the tuberculin test in the respective years.*

YEAR	CATTLE IN HERD	REACTORS	APPROCIMATE PERCENTAGE OF REACTORS
1924	30	0	—
1925	26	0	—
1926	30 to 40*	0	—
1927	30 to 40	1	—
1928	30 to 40	1	—
1929	40	0	—
1930	40	0	—
1931	48	3	6.25
1932	46	13	28.26
1933	60	2	3.33
1934	53	1	1.88

*Exact number unknown

METHODS

The material was brought to the laboratory within a few hours after the tissues had been removed from the carcasses and suitable portions were selected for the making of emulsions. After a preliminary treatment with 5 per cent oxalic acid for the purpose of eliminating non-acid-fast contaminants that might have been present, portions of the emulsified tissue were used to inoculate several tubes of Herrold's egg yolk-agar culture medium.[18] The balance of the emulsion usually was employed to inoculate two rabbits intravenously, and two guinea pigs subcutaneously.

If no growth occurred after three months, the cultures were considered negative; the guinea pigs were killed after eight weeks and the rabbits between ten to twelve weeks subsequent to injection. At the time of necropsy, the spleens of the respective guinea-pigs were emulsified and cultures attempted on Herrold's egg yolk-agar culture medium. Cultures were attempted also from any lesions that were encountered during necropsy of the respective rabbits.

The type, or identification of the respective strains of acid-fast bacilli that were obtained, was accomplished by the injection of the bacterial suspension into rabbits, guinea pigs and tuberculin-tested chickens.

TABLE II—*Summary of the inoculation of animals and of attempts to culture Mycobacterium tuberculosis from the tissues of eleven cows that had reacted positively to tuberculosis.*

Cow	Lesions	Tissue Examined	Laboratory Findings		
			Direct Culture	Animals*	Results
1	Congested bronchial lymph-node	Bronchial lymph-node	Negative	Negative	Negative
2	Few caseous foci in cervical lymph-node	Cervical lymph-node	Negative	Negative	Negative
3	Subcutaneous tuberculoid†	Tuberculoid lesions	Acid-fast bacilli	One guinea pig positive	*M. tuberculosis avium*‡
		Mesenteric lymph-node	Acid-fast bacilli	No injection	*M. tuberculosis avium*
4	Abscess of liver	Abscess of liver	Negative	Negative	Negative
5	Abscess of lung	Abscess of lung	Negative	Negative	Negative
		Bronchial lymph-node	Negative	Negative	Negative
6	Mediastinal lymph-node, congested	Mediastinal lymph-node	Negative	Negative	Negative
7	No visible lesions	Mesenteric lymph-node	Acid-fast bacilli	Negative	*M. tuberculosis avium*
8	Subcutaneous tuberculoid	Tuberculoid lesions	Negative	Negative	Negative
		Mesenteric lymph-node	Negative	Negative	Negative
9	No visible lesions	Mesenteric lymph-node	Negative	Negative	Negative
10	No visible lesions	Mesenteric lymph-node	Negative	Negative	Negative
11	Abscess in skin	Abscess in skin	Negative	One rabbit positive	*M. tuberculosis avium*
	Congested bronchial lymph-node	Bronchial lymph-node	Negative	Negative	Negative
		Mesenteric lymph-node	Negative	Negative	Negative

*In most instances, two rabbits and two guinea pigs received injections of the prepared inoculum.
†The term "subcutaneous tuberculoid" refers to the so-called "skin lesion."
‡The respective strains of acid-fast bacilli were designated as bacilli of avian tuberculosis after tests for pathogenity in which rabbits, guinea pigs and chickens were used.

MATERIAL STUDIED

Material from a total of eleven cows was subjected to laboratory investigation. This is summarized in table II. From the tissues of three of these animals (cows 3, 7 and 11) acid-fast bacillary forms, which were definitely identified as avian tubercle bacilli, were obtained. The pertinent facts concerning each of these animals follow:

Cow 3: This animal was born in the herd in 1927, and subjected to annual tuberculin tests. Following the injection of tuberculin on November 16, 1931, a reaction that was considered of a suspicious character was observed. The animal was retested in February, 1932, and while the reaction that occurred was somewhat atypical it seemed to be of sufficient magnitude and character to justify removal of the animal from the herd. The animal then was slaughtered. The necropsy disclosed the presence of two separate tuberculoid lesions in the subcutis of one front leg. One of these, specimen A, consisted of multiple abscesses, the necrotic contents of which were of a thick, creamy consistence and of a yellowish color. The other tuberculoid lesion, specimen B, was of a progressive, proliferative character and contained a few purulent foci. One of the mesenteric lymph-nodes appeared to be somewhat abnormal in appearance, although definitely characteristic evidence of tuberculosis was missing.

Acid-fast bacillary forms were isolated in direct culture from specimen A of the subcutis, and similar organisms were isolated from the spleen of one of the guinea pigs which was inoculated with portions of the emulsified lesion. Acid-fast bacilli were secured also by direct culture of the emulsion of the mesenteric lymph-node. Each of these strains proved to be pathogenic for rabbits and chickens but not for guinea pigs and each was therefore considered to have been derived originally from fowls.

Cow 7: This animal had belonged to the herd since 1929, having been born on the premises. She was tuberculin-tested in 1930 and 1931 and both tests were negative. There were two tests administered in 1932 and on both occasions a somewhat atypical reaction was obtained. Following the second test, the animal was sent to slaughter. At the necropsy no visible lesions were found. One of the mesenteric lymph-nodes was obtained for laboratory study. This was emulsified for the purpose of making cultures and for the inoculation of rabbits and guinea pigs. No lesions were observed when the respective animals were examined at necropsy, 90 days subsequent to injection. After rather prolonged incubation (93 days), a growth of acid-fast bacilli was obtained, which proved pathogenic for chickens and rabbits but not for guinea pigs.

Cow 11: The animal was born on the premises in 1931 and was tested with tuberculin as follows: once in 1931, twice in 1932, three times in 1933, and once in 1934. At the fourth test, both mammalian and avian tuberculin were administered. The reaction was slight but rather typical with mammalian tuberculin and a suspicious reaction was obtained with the avian tuberculin. The fifth test was adminstered 90 days later and typical reactions were obtained with both avian and mammalian tuberculins. Mammalian and avian tuberculins again were administered after intervals of 57 and 78 days, respectively, and typical reactions were obtained with both tuberculins. The animal then was slaughtered. The necropsy revealed a flattened, nodular mass about 1.5 cm in diameter in the subcutis of the lower extremity of the right hind leg. The lesion was dissimilar to the usual so-called "skin lesion." It proved to be an abscess which contained a grayish yellow,

purulent material in which acid-fast bacteria were not found on direct smear. There was also slight hyperemia of one of the bronchial lymph-nodes of the lung. No other abnormalities were noted.

The following material was utilized for the making of cultures and for the inoculation of rabbits and guinea pigs: purulent contents of the lesion of the subcutis; the bronchial lymph-node, and one mesenteric lymph-node. All of the culture attempts were negative, as were all of the guinea pigs and rabbits, with the exception of one rabbit, which had been injected with a portion of the emulsion prepared from the purulent contents of the abscess of the subcutis. This rabbit was killed 66 days after receiving the inoculum and at necropsy the spleen was found to contain numerous yellowish, miliary foci although the organ was enlarged very little if any. The other organs were non-tuberculous, both grossly and microscopically. A culture of acid-fast bacilli was obtained from the spleen of this rabbit. Subsequent observations pertaining to the pathogenicity of this strain disclosed that it was innocuous for guinea pigs, but markedly pathogenic for rabbits and chickens. In the first two passages through rabbits, after the strain was isolated, the lesions suggested an organism of substandard virulence for this animal, but after two passages through rabbits in succession, the organism exhibited the usual virulence of the avian form of *M. tuberculosis*.

Comment

This study has been of interest in offering a possible explanation for the sporadic occurrence of tuberculin-reacting cattle in a herd that has been subjected to tuberculin tests regularly during a period of eleven years, during which time none of the reacting animals have disclosed at necropsy definite or typical lesions of bovine tuberculosis. Since characteristic lesions of bovine tuberculosis were absent in 21 cattle, which had reacted to the tuberculin test, and which were subsequently examined at necropsy, it seems a reasonable assumption that the sensitivity that was demonstrated following the injection of tuberculin in these cattle was induced at least in some instances by acid-fast bacteria other than *M. tuberculosis bovis*.

Further evidence that the organism of avian tuberculosis may have been responsible for the tuberculin sensitivity of certain animals in this herd was the cohabitation of the cattle with chickens. Since the establishment of the herd, a flock of about 100 chickens has been maintained on this farm. The fowls have had the freedom of the premises and frequently feed in the barn and in the barnyard. Two and a half years ago, an attempt was made to tuberculin-test the flock but only about half of the

chickens could be caught. Tuberculin was administered to these and one reactor was found.

The chicken that reacted positively to tuberculin was a Brown Leghorn hen which had been on the premises for twelve years. The chicken was in excellent condition and the only lesion of tuberculosis found at necropsy was a nodular mass 0.6 cm in diameter, situated in the wall of the rectum 4 cm anterior to the vent. Microscopically, this was found to be composed of conglomerate tubercles, the majority disclosing central necrosis with numerous acid-fast bacteria in the cellular detritus.

While it is impossible to say what the actual incidence of tuberculosis may have been among the entire flock, it is not unlikely that the disease was present in a few if not many of the chickens that were not tuberculin-tested. Even though tuberculosis in this flock was limited to the one chicken that reacted to the test, the possibility for the widespread dissemination of the infective bacteria seems sufficiently great to account for its residence in the tissues of the three cows from which the organism was isolated. The enormous number of tubercle bacilli usually present in lesions of tuberculosis of chickens provide a condition favorable to the dissemination of the disease. This fact, together with the tendency of lesions to form along the walls of the intestines where they often ulcerate and continually discharge the infective bacteria into the lumen of the intestine, provides a circumstance that makes the tuberculous chicken a considerable menace to other live stock.

Summary and Conclusions

From a herd of dairy cattle that had been tuberculin-tested annually for a period of eleven years and in which there was cohabitation with tuberculous chickens, attempts were made by culture methods and the inoculation of laboratory animals to isolate the organism of tuberculosis from eleven of the animals that had reacted to tuberculin. In no instance was the bovine form of the tubercle bacillus obtained, although four strains of the organism of avian tuberculosis were isolated from three of the animals. From one animal, the organism was secured from a tuberculoid lesion in the subcutis and from a mesenteric lymph-node. From another animal the bacilli were obtained from a mesenteric lymph-node only, and in the third cow the infective organism was isolated from an abscess in the subcutaneous tissue.

These data confirm the findings of others concerning the occurence of the organism of avian tuberculosis in cattle and provide

additional evidence that the avian form of the tubercle bacillus may not infrequently sensitize cattle to both mammalian and avian tuberculins.

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1,000 Mules for Rehabilitation Work

When the Tennessee Emergency Relief Administration recently let a contract to purchase 1,000 mules to be used in rural rehabilitation work, Dr. M. Jacob, in his capacity of head of the Department of Animal Husbandry at the University of Tennessee, was asked to organize a system of inspection of the animals. Dr. Jacob accepted, with the understanding that he would be permitted to use veterinarians to make up the inspection organization. As a result, a number of Tennessee veterinarians will profit by the arrangement insisted upon by Dr. Jacob. No animal will be accepted unless it is pronounced fit by a member of the inspection force. The first consignment of 15 mules for Maury County arrived from Memphis on April 9, and the animals were inspected by Dr. R. B. Read (McK. '15), of Columbia, Tenn., inspecting officer for that section of the state.

STIFF-LAMB INVESTIGATIONS

Preliminary Report*

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For a number of years, sheep-raisers in Wyoming have reported losses from a condition which they call "stiff lambs." Such reports have come from various parts of the state. It was not until about two years ago that it was possible to do any direct experimental work upon the stiff-lamb condition that was being reported. In the spring of 1932, unusual losses were reported, and the junior author went into the field with considerable equipment, the purpose of which was the collection of pathological and bacteriological specimens for further study, as well as a detailed study of history, symptoms and lesions.

HISTORY

In those localities where this disease was encountered, owners reported losses from the same condition for a number of years. The trouble was more or less limited to farm flocks of from 50 to 500 head, where shed-lambing had been practiced. The same sheds and pens had been used for lambing over a period of years with little or no thought of sanitation. Treating the navel and asepsis at the time of docking and castrating had received practically no attention. Several owners reported that their losses from this condition could be controlled largely by postponing the lambing season until late in the spring, when the weather was sufficiently moderate to permit lambing in the open. Early season lambing, during February, March and the first part of April, usually confined the ewes to the barns because of weather, and the loss of lambs increased.

The number of lambs affected in the flocks where the stiff condition was encountered was found to average 15 per cent of all lambs dropped that year. The mortality was very high.

SYMPTOMS

The first objective symptoms were what appeared to be a stiffening of the muscles of one or more legs, possibly the result of arthritis. This might be accompanied by swellings at one or more articulations, particularly at the carpus or hock. The

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lambs appeared to experience difficulty in standing, either from pain or muscular inability, and lay down a large part of the time. In most cases, when attempting to walk they would move with a stilty gait, the feet being carried well under the body, so that it gave them a "humped" appearance. This condition would continue for considerable time without much change, or the lamb might gradually become worse, and in a period of 48 to 72 hours become unable to rise or able to do so only with considerable difficulty.

During this time the temperature remained normal. The pulse rate ranged from normal to as high as 200 per minute. The respiration rate varied from 40 to 70, with only slight exertion increasing this to as much as 150.

In many cases the feet became rather sharply flexed at the fetlock. Straightening might or might not cause a painful reaction. However, the feet practically always returned to the same flexed position when released.

The appetite remained fairly good. If bottle-fed or held up to the ewe, the lambs would suckle vigorously and live for long periods of time, some of them even recovering sufficiently to walk again. Otherwise the ewes would forsake them, soon drying up, and the lambs would gradually starve to death. Constipation was a rather constant symptom.

Gross Lesions

The gross lesions that were seen with any constancy and that were of any particular significance were found in the joints, skeletal muscles, heart and kidneys.

In most cases one or more joints showed purulent arthritis. There were a few cases in which arthritis existed but pus was not present. In these the articular cartilages contained hemorrhages.

In some cases the skeletal muscles showed white lesions. These had a somewhat similar gross appearance to those described as white-muscle lesions by other workers. The appearance was somewhat like albuminous degeneration or beginning coagulation necrosis. Only parts of the muscles were affected. A longitudinal strip of a single muscle, usually round and running for varying lengths of the muscle, appeared to be the chief change. These areas varied in diameter from 1 to 30 millimeters. The length varied from a few millimeters to the entire length of the muscle. The occurrence of the white-muscle lesions on opposite sides of the body was fairly constant, although the extent of the

affection was considerably different. The regions of the body in which white-muscle lesions were found were the heavy muscular regions of the shoulder, arm, rump and thigh. Purulent arthritis also was usually found in the cases showing white-muscle lesions.

Lesions of the heart were usually present. Hypertrophy and albuminous degeneration were common. Frequently white areas were found in the muscle tissue beneath the endocardium. Such whitish lesions in the heart muscle were found to be of different histological structure than the white lesions of the skeletal muscles. These white lesions in the myocardium were about 1 mm wide and 4 to 8 mm in length. Small abscesses 3 or 4 mm in diameter were found also in various parts of the heart in some cases.

The kidneys often had subcapsular ecchymotic hemorrhages. Abscesses of from 1 to 15 mm in diameter also were found in the cortex in some cases.

Collection of Materials

Material was collected from carefully autopsied cases which had just been killed. As a routine practice the tissues were collected and placed in Zenker's fluid and also in 10 per cent formalin. The tissues thus collected were from the skeletal muscles, heart, kidneys, nerves, spinal cord, cerebrum, cerebellum, medulla oblongata, hippocampus, lungs, liver, spleen, abomasum and small intestines. Cooked meat medium and double-strength, beef-infusion agar slants were inoculated with heart-blood, spinal and joint fluid. In addition to these specimens, three typically sick animals were brought back to the main laboratory and were later used in the same way.

Pathological Histology

All tissues were negative for consistent and what appeared to be significant microscopical lesions except the white skeletal muscles, heart and kidneys.

The white-muscle lesions were quite similar to those reported by various authors for white-muscle disease of lambs. These showed fibroplastic tissue to a moderate extent. In addition the fibers showed the sarcolemma ruptured in many places. There were areas of pronounced leukocytic infiltration, probably neutrophilic and lymphocytic, but granulocytes seemed to predominate. Large numbers of eosinophiles were seen in a few sections. These infiltrating cells were seen in both the ruptured and unruptured fibers. Some ruptured fibers showed granules as seen in albuminous degeneration.

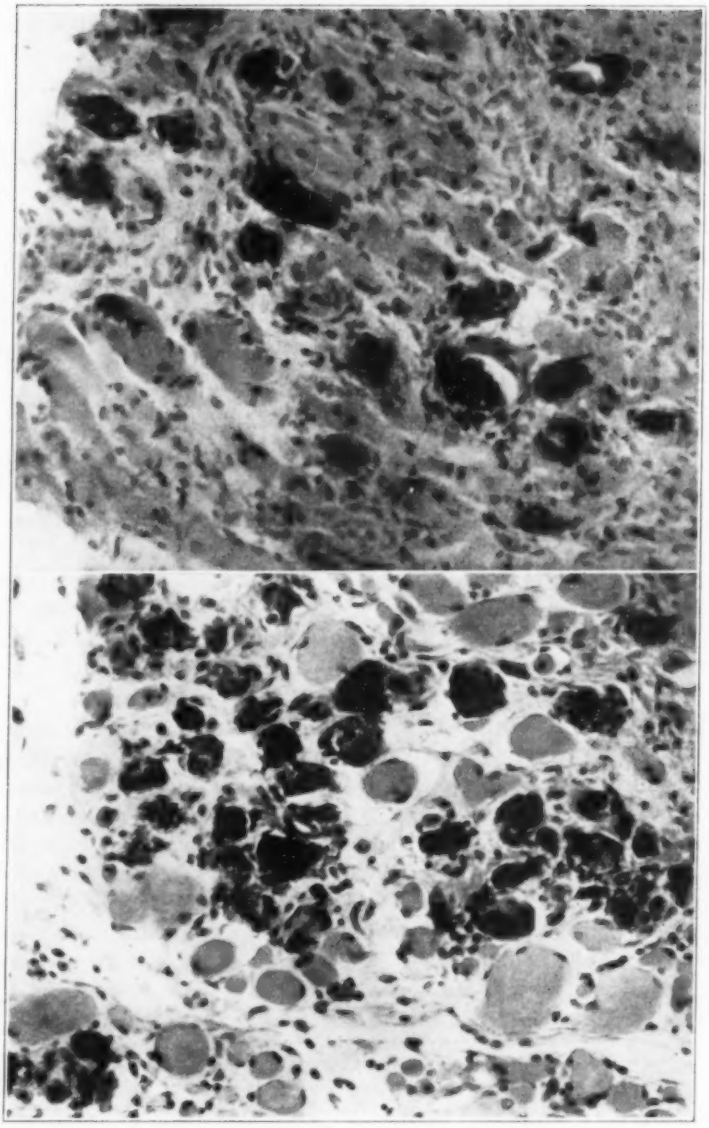

FIG. 1 (above). White-muscle lesion lamb 10 (x 200).
FIG. 2 (below). White-muscle lesion lamb 11 (x 200).

There were also present quite uniformly in the white-muscle lesions depositions of hemotoxylin-staining substances within the broken-down muscle fibers. Some apparently were excessive cellular infiltrations, but others looked like salt deposits. These overstaining areas were uniform throughout sections stained at various times and did not look like faulty staining with hemotoxylin.

Chemical examination of these white-muscle lesions compared with unaffected lamb muscle showed a great increase in the amount of inorganic salts present.

The heart and kidneys showed lesions one would expect in navel infection, infection of docking wounds, etc., as have been reported by authors in their experiments upon some forms of arthritis in lambs. These consisted of abscesses, streaks of coagulation necrosis, and small circumscribed areas of intensive leukocytic infiltrations, probably starting abscesses.

Bacteriological Work

Blood-smears and smears of the spinal fluid were negative microscopically. Cooked meat media and agar slants inoculated with a loopful of blood and of spinal fluid failed to show any growth. However, when one or two cubic centimeters of blood was aseptically removed from the living cases and from cases as soon as killed and placed in cooked meat medium, growth was obtained. In many cases, these were definitely proved to be pure cultures. The pathogenic organisms so isolated could be placed in one of three groups according to their morphology.

One of these groups consisted of short, bipolar, Gram-negative, non-spore-forming, rod-shaped organisms that produced acid and gas on dextrose, lactose, maltose and mannite, but did not produce gas or acid on sucrose (fig. 3). Another group consisted of longer, Gram-negative rods that did not have bipolar appearance, were non-spore-formers, and had the same fermentation reactions on the five sugars named above. The third group were spherical organisms with irregular arrangement and were Gram-positive. They produced acid but no gas on the same five sugars. No other work has been done to identify these organisms, but animal inoculations into lambs and rabbits have been made.

Animal Inoculation

Pure cultures isolated were first injected subcutaneously into lambs, rabbits and guinea pigs. The only results were local lesions at the site of injection. Intravenous injections were then made into rabbits and lambs. The results of injections of the bipolar group are shown in table I. Very similar results were

TABLE I—*Intravenous injection of the group of bipolar organisms.*

ANIMAL	CULTURE	AMOUNT (cc)	KIND OF CULTURE	DATE OF INJECTION	RESULTS
Rabbit 18	3–1	1.0	Cooked meat	June 29	Stiff July 1. Dead July 3. Pus in joints
Rabbit 27	3–1	1.25	Agar slant	July 6	Dead July 9. Negative findings
Lamb 2019	3–1	3.0	Agar slant	Aug. 16	Dead Aug. 17. Negative findings
Rabbit 21	3a–1	1.0	Cooked meat	June 30	Stiffness and incoordination for 15 days. Recovered. Dead Oct. 24
Lamb 2018	3a–1	3.5	Agar slant	July 12	Stiff and depressed July 15. Dead July 20. Pus in joints
Rabbit 30	9–1	1.0	Agar slant	July 18	Became very sick. Died July 20. Negative findings
Rabbit 28	9–4	1.25	Agar slant	July 6	Little stiff July 21. Dead July 25. Pus in joints. White-muscle lesions
Rabbit 34	11a–1	1.75	Agar slant	Aug. 3	Partial paralysis of legs. Died Dec. 1. Negative findings
Rabbit 23	2012–1	0.5	Cooked meat	June 20	Stiff July 5. Paralyzed July 21. Dead July 27. White-muscle lesions
Rabbit 24	2012–1	1.5	Agar slant	July 2	Stiff July 5. Dead July 14. Pneumonia
Rabbit 43	2012–1	1.0	Agar slant	Aug. 16	Sick and depressed Aug. 18. Dead Aug. 24. Pus in joints
Lamb 1286	2012–1	4.0	Cooked meat	July 12	Little stiff for week. Recovered
		4.0	Agar slant	Aug. 16	Dead Aug. 17. Negative findings

obtained by the two other groups, with the exception that no white-muscle lesions were produced with the other rod form.

Clinical symptoms and postmortem lesions in general, as seen in the lamb field cases, were reproduced in lambs and rabbits injected intravenously with the three types of organisms isolated in pure cultures. White-muscle lesions were found in some of the rabbits infected with two of these three types of organisms. The organisms were isolated again from the lambs and rabbits inoculated experimentally.

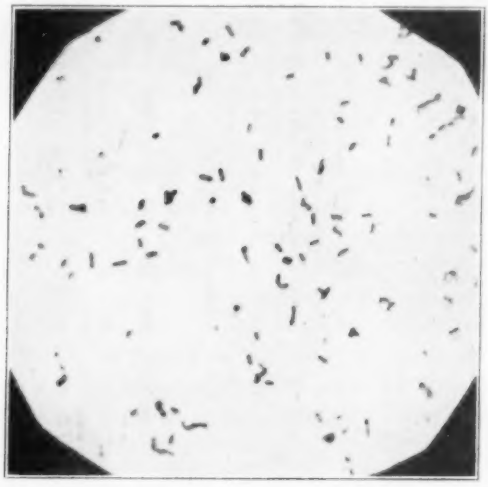

FIG. 3. Bipolar organism (x 1300).

LITERATURE AND DISCUSSION

Stiff-lamb conditions have been reported and investigated by a number of research workers. Most of these workers have reported what sheepmen call "stiff-lamb disease" under one of three conditions, namely, white-muscle disease, specific arthritis, and non-specific arthritis. It is not the contention in this paper that white-muscle disease is the same as either the specific or the non-specific arthritis. The work showing occasional white-muscle lesions apparently produced by cultures isolated from field cases seemed interesting and is reported for this reason.

The most extensive work reported upon stiff-lamb disease from the white-muscle disease aspect as a separate condition from any of the various forms of arthritis has been by Cornell University. Metzger and Hagan[1] found a stiff-lamb condition quite

prevalent in certain sections of New York. White-muscle lesions were consistently present. Their gross and microscopic appearance seemed to be identical with the white-muscle lesions encountered in Wyoming. Symptoms in the report were quite similar to those described above. Bacteriological findings were negative. Arthritis was reported in one lamb which had white-muscle lesions. They found heart lesions inconsistently present. These consisted of whitish patches which showed degeneration and fibroplastic tissue. They believed the condition was a specific disease of an azoturia-like nature, in which the diet and exercise were factors.

Willman *et al.*[2] worked upon the same condition as a continuation of the work of Metzger and Hagan. They made no bacteriological examinations. They were able to reproduce a variable percentage of stiff lambs with white-muscle lesions by feeding oats, barley, cull beans, and second-cutting alfalfa hay. Exercise did not seem to be a factor in reproducing the disease, as lambs on certain rations developed stiffness when given exercise and when not given exercise, while lambs on other rations would develop no stiffness, regardless of whether given exercise or not. They were unable to reproduce stiffness and white-muscle lesions in rabbits with the same ration fed to the lambs that developed the condition. In their discussion of the possible causes they suggested that the high quantity or the poor quality of the protein may be the cause.

Marsh[3] reported a stiff-lamb, white-muscle disease in Montana. The gross and microscopic appearances of these white-muscle lesions were apparently the same as those encountered in field cases in Wyoming and also reproduced by injections of cultures. He suggested a disturbance in the phosphorus-calcium balance as a possible cause, as chemical analysis of the white-muscle lesions showed 12 to 15 times more calcium phosphate than was found in normal lamb muscle.[4] Chemical analysis of white-muscle lesions found in Wyoming showed 22 times as much calcium and four times as much phosphorus as found in apparently normal lamb muscle.

Jungherr and Welch[5] also reported upon apparently the same white-muscle condition in Montana, and found the frequent presence of a diplococcus in the heart-blood and coccidia in the intestines of many of their white-muscle, stiff-lamb cases.

White-muscle disease in lambs has been reported in several other states. Hobmaier[6] reported a similar condition in Germany. Marsh[3, 4] reported a non-specific infection as the cause of

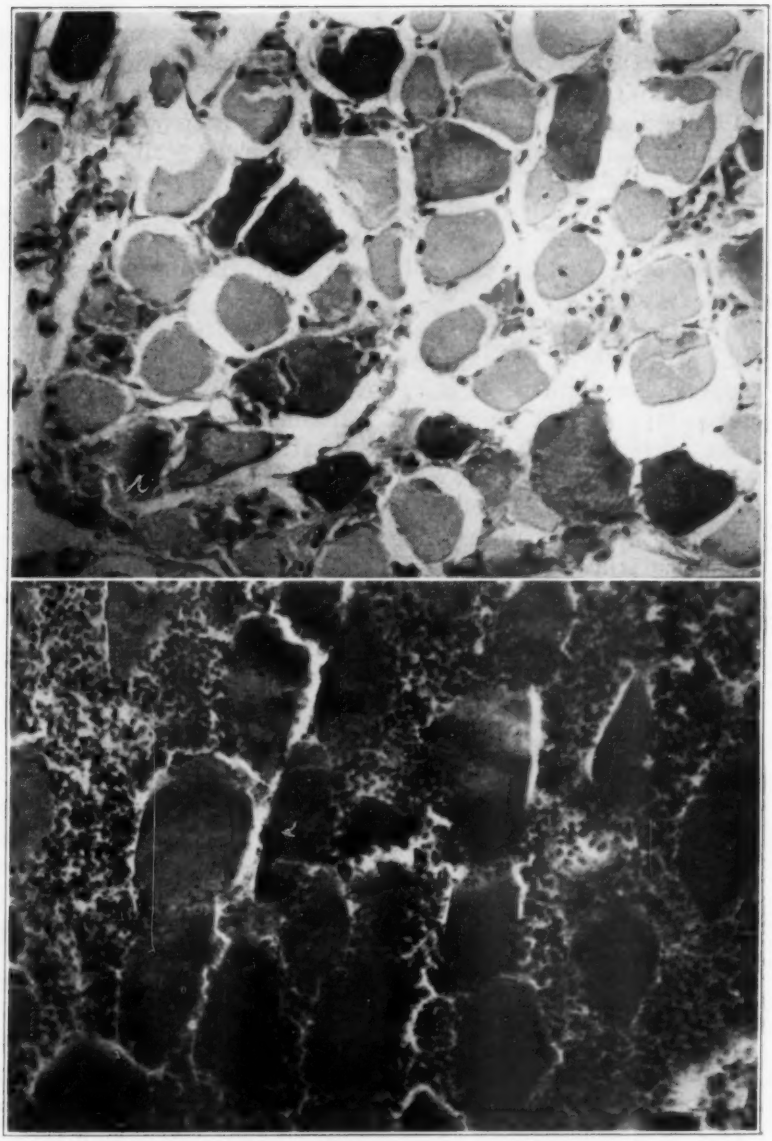

FIG. 4 (above). White muscle lesion rabbit 22 (x 200).
FIG. 5 (below). White-muscle lesion rabbit 23 (x 200).

arthritis in lambs with enlargement of the joints and the presence of pus. No white-muscle lesions coexisted. The same author[7, 8] reported a specific infection of lambs with *Erysipelothrix rhusiopathiae*. He was able to reproduce the disease. In this condition he found no visible enlargement of the joints during life. There was no pus present, and white-muscle lesions did not exist. The organisms isolated by the authors from white-muscle lesion cases with purulent arthritis were not *E. rhusiopathiae*. Howarth[9] gives a detailed report of specific arthritis in California due to *E. rhusiopathiae*. Kinsley[10] and many others have reported arthritis in lambs from which pyogenic staphylococci were isolated from the purulent joint fluid. Ray[11] found *E. rhusiopathiae* as the cause of arthritis in lambs in Missouri.

Tanaka[12] injected rabbits, dogs and guinea pigs intravenously, as well as by way of the body cavities, with calcium salts in doses up to 6 grams. His results showed deposits of calcium phosphate and calcium carbonate with special frequency in the striated muscle.

Table II shows the ration of the ewes, of which the lambs were eating a very small amount. The milk did not appear to be a factor, as never more than one of twins showed stiffness. There is nothing in the ration in the nature of too heavy concentrates. Also, the appearance of the condition under so many different rations would seemingly eliminate a disturbance in the calcium-phosphorus balance as the cause in these cases. The rabbits that were injected with cultures obtained from the heart-blood of the field cases were receiving a ration of alfalfa hay, bran, oats and corn. White-muscle lesions and stiffness were not encountered in any rabbits not injected, whereas a few injected developed white-muscle lesions.

The authors feel confident that the cause of the stiff-lamb conditions encountered so far in Wyoming is the infection of the navel and the docking and castration wounds with the bipolar rod, staphylococcus, and the third group of rod-shaped organisms reported here. No doubt there are other organisms in the same or other localities which were not isolated. It appears that white-muscle lesions may sometimes develop in a small percentage of stiff lambs where the stiffness is due to bacterial infection. Ordinary smears and inoculations were negative, and 1 to 2 cc of the heart-blood was used as inoculating material. Otherwise bacteriological findings except in the purulent joints would have been negative. It seems reasonable that infection may sometimes produce a disturbance in the physiological function and chemical

TABLE II—*Data on lambs and rations of ewes.*

FLOCK	LAMBS	STIFF LAMBS	DOCKED OR CASTRATED	AGE OF LAMBS AFFECTED	FEED EWES WERE GETTING
1	105	32	No	7–31 days	First, second cutting alfalfa hay. Little oat hay to some
2	65	3	Docked 3–4 days before symptoms appeared	3–4 weeks	First cutting alfalfa hay
3	80	5	No	1–2 weeks	Sudan and timothy
4	120	15	Both during height of trouble	3–4 weeks	First, second cutting alfalfa hay
5	400	22*	Both during height of trouble	2–3 weeks	Alfalfa, timothy, oat hay until lambing, then second cutting alfalfa hay
6	50	2	Both. Symptoms appeared one week later	2–3 weeks	Alfalfa hay and oats
7	58	20	Both. Symptoms appeared one week or ten days later	2–4 weeks	First cutting alfalfa until lambing, then second cutting alfalfa hay
8	93	12	Both during height of trouble	2–3 weeks	Native hay until lambing, then first cutting alfalfa hay

*Lost 33 others not typically stiff.

activity with the subsequent deposit of salts in the muscle. Wells[13] offers several hypotheses explaining the chemistry of the progress of calcification which might also be used here.

SUMMARY

1. A study of the history, symptoms, gross and microscopic lesions, and bacteriological findings has been made of a condition in Wyoming which sheepmen call "stiff lambs."

2. Gross lesions encountered have been somewhat variable. These have consisted of arthritis, white skeletal muscle lesions, greyish white areas and streaks in the heart and in the kidneys.

3. Microscopical examination of the white skeletal muscle lesions showed degeneration, cellular and calcium salt infiltrations. The heart and kidney lesions were found to be areas of coagulation necrosis and purulent foci.

4. Chemical examination of the white-muscle lesions showed an increase in the calcium and phosphorus present.

5. When large quantities of the heart-blood were used for inoculating media, cultures were obtained.

6. The significant organisms isolated were divided into three groups according to their morphological characteristics. These were bipolar rods, cocci, and non-spore-forming, rod-shaped organisms.

7. All three of these different forms of organisms reproduced typical symptoms in lambs and rabbits when injected intravenously. Two of these three forms reproduced white-muscle lesions in rabbits. Purulent arthritis was the most common lesion reproduced in both rabbits and lambs by these pure cultures.

8. The history showed that the navel was untreated and that a hot docking instrument was not used.

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TREATMENTS FOR CYANIDE POISONING OF SHEEP AND CATTLE*

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There exists in the United States a number of cyanogenetic forage plants of wide geographical distribution, which create a nation-wide problem of cyanide poisoning of live stock.[1] The tendency of these plants to develop increased amounts of prussic acid when grown under drouth, or some other unfavorable condition, has of late tended to accentuate this problem.

Previous workers have demonstrated the efficacy of certain drugs against cyanide poisoning, by tests conducted with small laboratory animals. Fitch and his coworkers in Minnesota (personal communication) have developed significant remedial results using sheep and cattle as experiment animals. Opportunities have been afforded workers in the Bureau of Animal Industry to test the practical value of several remedies when applied to cattle and sheep. It is the purpose of this paper to review briefly the salient findings of our investigations, the complete details of which have been reported elsewhere.[2-4]

The cyanide was administered to our experiment animals orally in the form of a freshly prepared solution of potassium cyanide and, with two exceptions, the remedies were given intravenously or intraperitoneally.

SYMPTOMS OF CYANIDE POISONING

Symptoms occurred from 0.5 to 2.5 minutes after completing the drench. The first symptom usually was an increase in rapidity and depth of respiration. Other symptoms which followed in cattle were increased pulse rate, hypersensitivity to stimuli, and spasmodic muscular movements. More severely poisoned cases showed a rapid increase and deepening of respiration, then respiratory convulsions and dyspnea. These developed on an average of three minutes after the administration of the cyanide.

In sheep, convulsions of varying degree and duration occurred in about two-thirds of the cases, consisting of violent kicking, opisthotonos, clonic muscular spasms and jerky movements of the eyeball. In cattle the jugular vein was prominent and engorged during this period and venous blood was bright red. Meanwhile respirations became dyspneic and retarded. In

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sheep which recovered, dyspnea lasted from one to three minutes and the animals gradually improved. In fatal cases dyspnea lasted for an average of 17 minutes.

During dyspnea the respiratory rate varied, ranging from panting to spasmodic gasping. In some cases it was deep and irregular, with forced expirations. Mild cases gradually recovered. In fatal cases respiration became increasingly shallow, with lengthening intervals until life was extinct. Cyanosis was more or less general during this final stage, and the animal remained in a comatose condition. The eye reflex was apparently normal until the end.

Cattle receiving sublethal doses of cyanide without treatment manifested relatively rapid recuperative powers. Sheep varied in time of recovery from several minutes to as many hours. Much variation in time of sickness existed for both cattle and sheep receiving lethal doses without treatment.

Several cows dying of cyanide poisoning were autopsied. No constant lesions of the poisoning were observed, however, the more common characteristics being dark muscle tissues, congestion or hemorrhage of lungs, petechiation of tracheal mucosa, and a frothy, bloody discharge from the mouth which last occurred in two cases at death.

Determination of Lethal Dose of Hydrocyanic Acid

In order to appraise the value of therapeutic measures, on experimentally poisoned cattle and sheep, it was first necessary to determine the lethal doses of cyanide for these species. Various amounts of potassium cyanide were tried, and the lethal dose for cattle, calculated as hydrocyanic acid, was determined to be nearly 2.042 mg per kilogram of body weight. That for sheep was determined to be 2.315 mg per kilogram of body weight.

Experiments with Remedies

Several substances were next tried, either singly or in combination, for the treatment of cyanide poisoning. These included methylene blue, sodium thiosulfate, sodium tetrathionate, and sodium nitrite.

Methylene blue: In sheep it was found that 50 cc of a 1 per cent methylene blue solution administered intraperitoneally 4.5 minutes after the oral administration of 1.42 minimum lethal doses of the cyanide resulted in the recovery of the animal. In this species 30 cc of the methylene blue solution failed to protect against the same dose of cyanide.

Sodium tetrathionate: When administered *per os* simultaneously with 1.42 m.l.d. of cyanide, 10 cc of a 10 per cent solution of sodium tetrathionate enabled a sheep to recover after transient symptoms of cyanide poisoning. Other cases receiving 20 cc or 30 cc of the sodium tetrathionate solution intraperitoneally following the cyanide succumbed to the poisoning.

Sodium thiosulfate: A cow receiving 1.39 m.l.d. of cyanide recovered following the intravenous injection of 20 cc of a 10 per cent solution of sodium thiosulfate. Intraperitoneal injection of 20 cc of this solution in sheep protected against 1.66 m.l.d. of cyanide.

This remedy possesses definite value even when unaided by combination with other agencies, being tolerated in reasonably large amounts without deleterious effects on sheep or cattle. Sodium thiosulfate is identical with photographers' "hypo," often miscalled "sodium hyposulfite." The technical grade of photographers' "hypo" may, in emergency, be employed in treating cattle or sheep for cyanide poisoning.

Sodium nitrite: Administered intravenously, to cattle, within ten minutes after the cyanide, 20 cc of a 10 per cent solution of sodium nitrite was shown to impart protection against one lethal dose, but this amount was ineffective against 1.5 lethal doses of the cyanide, when given 21 minutes after poisoning.

When given orally or intraperitoneally to sheep, 10 cc of a 10 per cent solution of sodium nitrite protected against 1.42 lethal doses but not against 1.66 lethal doses of cyanide.

Sodium thiosulfate and sodium nitrite combined: These two drugs were employed simultaneously in certain cases on the ground that they exerted different therapeutic influences on cyanide poisoning. Cattle having received up to 2.0 lethal doses of the cyanide recovered when given 10 cc of a 20 per cent solution of sodium nitrite and immediately afterwards 30 cc of a 20 per cent sodium thiosulfate solution intravenously within 4.5 minutes after the poison. The combination failed, however, against 2.5, 3, or 4 lethal doses of cyanide even when administered more promptly.

In view of the apparent superiority of this combination of remedies over the other remedies previously discussed, this treatment was tried on a number of sheep given from 2.0 to 3.5 lethal doses of cyanide. It was found that if the remedy was administered promptly, at least before the onset of gasping symptoms, sheep recovered from cyanide poisoning when given 10 cc of a 10 per cent solution of sodium nitrite, immediately followed

by 20 cc of a 10 per cent solution of sodium thiosulfate, both intraperitoneally. The combination treatment was effective against 2.75 lethal doses but not against 3.0 or 3.5 lethal doses of the cyanide in sheep.

In cyanide poisoning the symptoms are a direct result of the action of the poison on the central nervous system, first stimulating and then depressing. The course of these influences is most readily observed on the respiration and pulse rate, which are at first accelerated and later retarded and irregular. A similar sequence of symptoms follows the action of the cyanide on the motor centers. In fatal cases death is usually due to asphyxia as a result of paralysis of the respiratory center. Clonic convulsions of asphyxial origin are usually manifested in extreme cases. At death the heart continues to beat for a brief period following the cessation of respiration.

The principle of the nitrite-thiosulfate antidote for cyanide poisoning is a dual one. It is known that sodium nitrite converts hemoglobin into methemoglobin in the red corpuscles, and that methemoglobin has a strong affinity for hydrocyanic acid, forming a stable compound, cyanomethemoglobin. The hydrocyanic acid thus bound is unable to exert a toxic action upon the nerve centers. The action of sodium thiosulfate upon the cyanide is more direct, resulting in the formation of the relatively nontoxic sodium thiocyanate.

Limits of Time Between Cyanide Poisoning and Effective Treatment

Inasmuch as the interval of time between administration of the cyanide and the treatment appeared to be a determining factor, with regard to the recovery or death of the animal, a series of tests was made to establish how soon after the cyanide was given the remedy must be injected in order that the animal might survive.

Fifteen sheep were employed in the experiment. Each received an oral dose of cyanide equivalent to 1.5 minimum lethal doses. The remedy in each case consisted of 10 per cent aqueous solutions containing 1 gram of sodium nitrite and 2 grams of sodium thiosulfate respectively, and these were administered intraperitoneally at varying intervals after the poison. The nitrite and thiosulfate solutions may, however, with equal effectiveness be given as a combined injection. Animals which were given the treatment within four minutes after receiving the cyanide recovered. Those from which the remedy was withheld for longer intervals died, except for three atypical cases.

Four other sheep receiving a similar dosage of cyanide were given intraperitoneal injections of 0.5 gram of methylene blue, at respective intervals of four to seven minutes after poisoning, but these all died.

Determination of Therapeutic Limits of Sodium Nitrite

Tests were made to determine the degree of toxicity of sodium nitrite, as a gauge on the therapeutic limits of this substance. Much variation, however, was manifested in the effects of different doses given intraperitoneally, resulting in the conclusion that the safe limit for therapeutic use of sodium nitrite against cyanide poisoning is 1 gram intraperitoneally for a 75- to 100-pound sheep. For cattle the safe therapeutic dose of sodium nitrite was concluded to be from 2 to 3 grams.

Cyanide Poisoning a Veterinary Problem

The treatment of cyanide poisoning in sheep and cattle is distinctly a veterinary problem, and the practitioner in affected parts of the country will want to be informed and in readiness for this class of cases, which may be thrust upon him without previous warning and which will call for vigorous and undelayed action on his part. Having diagnosed the manifested case or cases as cyanide poisoning, his first effort will be to administer the treatment, which, if given in time, may at least save some of these cases. No time, however, should be lost in administering the treatment to other animals exposed to the poisonous forage, but not yet manifesting symptoms of cyanide poisoning. In fact, this latter duty may, in the judgment of the veterinarian, sometimes take precedence over the former one, depending upon conditions obtaining on his arrival.

The recommended treatment for sheep is the intraperitoneal injection of 1 gram of sodium nitrite and 2 to 4 grams of sodium thiosulfate, both in 10 per cent aqueous solutions, and for cattle, the intravenous injection of 10 per cent aqueous solutions containing 2 to 3 grams of sodium nitrite and 10 to 20 grams of sodium thiosulfate. For either animal the injection of sodium thiosulfate may be repeated if deemed advisable by the veterinarian, but in no case should additional amounts of sodium nitrite be given, as the original dose approximates the safe therapeutic limits of that drug.

Solutions of sodium nitrite and sodium thiosulfate are quite stable, and may be sterilized by boiling without any appreciable alteration of therapeutic value. The drugs are everywhere com-

mercially available and inexpensive. It therefore seems entirely possible for veterinarians in cyanide-affected areas to prepare themselves easily against this possible emergency by the preparation and sterilization of an adequate supply of these solutions.

SUMMARY

The problem of cyanide poisoning of live stock on range is geographically widespread.

The general symptoms of experimental cyanide poisoning are respiratory stimulation and accelerated pulse, later prostration and clonic muscular spasms, respiratory convulsions and dyspnea, terminating in coma and death.

Postmortem lesions of experimental cyanide poisoning in cattle are variable, the most constant being darkened musculature, pulmonary congestion and hemorrhage, and petechiation of tracheal mucosa.

The lethal dose of hydrocyanic acid for cattle is approximately 2.042 mg per kilogram body weight and that for sheep is 2.315 mg per kilogram body weight.

In the treatment of cyanide poisoning, methylene blue, sodium tetrathionate, sodium nitrite, and sodium thiosulfate, when used independently as intraperitoneal injections in sheep or intravenous injections in cattle, were found to possess some therapeutic properties. Solutions of a combination of sodium nitrite and sodium thiosulfate given in the manner stated proved more efficacious, and this was deemed the most satisfactory of all treatments tried. This protected against 2 m.l.d. in cattle and 2.75 m.l.d. in sheep.

Therapeutic doses of sodium nitrite and sodium thiosulfate solutions protected against 1.5 m.l.d. of cyanide in sheep when administered within four minutes after poisoning.

The limit of tolerance for sodium nitrite is 1 gram for sheep (75 to 100 pounds body weight) and 2 to 3 grams for cattle.

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MOSQUITO TRANSMISSION OF EQUINE ENCEPHALOMYELITIS*

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The epizoölogy of equine encephalomyelitis strongly suggests an active insect vector. Kelser[1] was first to report successful insect transmission of this disease. He recorded the transmission of equine encephalomyelitis from diseased to healthy guinea pigs and from diseased guinea pigs to a healthy horse by means of the yellow fever mosquito, *Aedes aegypti* (L.). Merrill, Lacaillade and TenBroeck[2] reported the transmission of both the eastern and western strains of equine encephalomyelitis by means of the salt marsh mosquito, *Aedes sollicitans* (Walker). This mosquito was first able to transmit the disease seven days after feeding upon virus suspension of guinea pig brain, eleven days after feeding upon diseased guinea pigs, and 20 days after being fed upon a diseased horse. Transmission with *Aedes cantator* (Coq.) and *Aedes aegypti* (L.) also was reported.

Studies conducted at the Utah Agricultural Experiment Station since September 1, 1933, indicate that two species of native Utah mosquitoes are capable of transmitting the western virus strain of equine encephalomyelitis from diseased to healthy guinea pigs. The species implicated are *Aedes nigromaculis* (Ldw.)§ and *Aedes dorsalis* (Mg.). As far as the writers are aware, the literature does not record transmission of western virus by these species.

Methods of collecting, handling and feeding the mosquitoes are to be reported elsewhere.[3] Guinea pigs inoculated with virus were placed in gauze bags and laid upon the tops of the gauze-covered mosquito cages. The refeeding on healthy pigs was accomplished in the same manner, except that those mosquitoes observed not to fill with blood were placed in celluloid cylinders (gauze fastened over the ends) and the cylinder end placed on the clipped belly of a guinea pig. The virus used in these studies was obtained from horses affected in the 1934 outbreak in Cache County, Utah. The virus was found to be identical with the so-called "western" type which has been reported in western areas of this country. Guinea pigs injected with virus emulsion usually received 0.2 cc of a 2 per cent brain emulsion, given intracranially.

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§Mr. J. A. Rowe has called the writers' attention to the presence, among the material used as *Aedes nigromaculis*, of specimens which closely resemble, and may actually be, *A. sollicitans* (the New Jersey salt-marsh mosquito).

Guinea pig brains were removed either at the height of the febrile reaction or after symptoms developed, following which they were preserved in 50 per cent buffered glycerin solution, pH 7.3 to 7.6.

Case 1: Approximately 200 *Aedes nigromaculis* collected as adults in meadows west of Logan were allowed to feed upon five guinea pigs which had previously received intracranial injections of virus emulsion (table I). These mosquitoes were then allowed to refeed on guinea pig 570 after three days had elapsed following their infective feeding. The results were negative. After seven days had elapsed following the infective feeding, the mosquitoes were fed on healthy guinea pig 505. This pig was prostrate two days after exposure, but subsequent trials showed no active virus present. After eleven days had elapsed following the infective feeding, the mosquitoes were fed on guinea pig 607. These feedings were continued on the 15th, 16th, 18th, 20th and 22nd days. Soon after this, all mosquitoes had succumbed. This pig was found prostrate 13 days following its first exposure to infective mosquitoes (two days following its last exposure). The brain of this pig (607) was in turn emulsified and used to infect other pigs. After three passages were made through a series of guinea pigs, the emulsion was passed through a Berkefeld N filter; it continued to be infective for two further guinea pig passages. A horse was then exposed intracranially to 8 cc of this virus. On the first day following inoculation, the horse ap-

TABLE I—*Data on guinea pigs which provided infective feeding for mosquitoes.*

CASE	GUINEA PIG	VIRUS	TEMPERATURE AT FEEDING (F. °)	INTERVAL BETWEEN INJECTION AND FEEDING (DAYS)	OUTCOME
1	284	W*	104.0	4	Recovered
	519	A*	102.2	6	Destroyed 14th day
	421	C	103.6	5	Died 5th day
	543	S	104.0	1	Died 4th day
	545	C*	104.4	1	Destroyed 4th day
2	509	F*	103.2	7	Recovered
	502	M*	104.8	7	Died 8th day
	543	S	103.2	2	Died 4th day
	545	C	104.2	2	Destroyed 4th day
3	172	S	104.3	4	Destroyed 4th day
	107	S	104.1	4	Destroyed 4th day

*On subsequent tests, the brain emulsion from these pigs did not prove to contain active virus.

peared somewhat listless but on the second day assumed a more normal attitude. On the third day, frequent yawning, grinding of teeth, and irritability were manifested. On the fourth day, the horse backed into a corner, fore limbs pushed forward; occasionally it crossed its posterior limbs. Teeth grinding and yawning continued and a sweetish putrid odor emanated from the mouth. The fifth day found the horse prostrate. Temperatures varied between 101.4 and 102° F. On this day, the horse was destroyed and its brain tissue preserved. Control guinea pigs, inoculated with the same emulsion received by the horse, were destroyed on the fourth and fifth days, with characteristic records of high temperature and loss of weight. Another series of guinea pigs, injected with emulsion prepared from the brain stem of the horse which had been destroyed, showed a temperature and weight chart characteristic of equine encephalomyelitis.

Case 2: Approximately 200 *Aedes nigromaculis*, collected in the meadows west of Logan, were allowed to feed upon the injected guinea pigs as listed in table I. It will be noted that two of the pigs used had also been used in case 1, the only difference in conditions being that the pigs were one day further advanced with the disease. It should be noted also that they were the only two pigs used in this case which were shown to carry virus, as proved by later tests.

After six days had elapsed following the infective feeding, the mosquitoes were fed upon healthy guinea pig 581. These feedings were continued on the 13th, 15th and 17th days, by which time all mosquitoes had succumbed. This pig (581) was found paralyzed 22 days following its first exposure and five days following its last exposure to infective mosquitoes. The brain was emulsified and further injections made into other pigs until three such passages were completed, two or more animals always being used in each trial passage. One passage included passing the virus through a Berkefeld N filter. One of three pigs injected with filtrate developed the characteristic disease course but succumbed before the brain could be harvested for further tests. A horse was then exposed, intracranially, to 6 cc of this line of virus; a series of guinea pigs serving as controls were inoculated at the same time. A high percentage of the pigs showed characteristic temperature and weight loss and were destroyed.

The horse appeared normal for the first two days following inoculation. On the third day, there was a tendency to grind the teeth. On the fourth day, there was an inclination to turn to the right. The fifth day found the horse still able to eat but showing incoördinated movements, particularly with the rear limbs; the

temperature was 102° F. On the sixth day, the horse was prostrate, showed twitching of the labia, icterous sclera, fetid odor from the mouth, and a temperature of 104° F. The horse was destroyed and the brain preserved for future tests. Subsequent inoculation of brain stem emulsions from this horse readily produced the disease in guinea pigs.

Case 3: Approximately 300 *Aedes dorsalis* were allowed to feed upon guinea pigs previously infected by intracranial virus injections (table I). These mosquitoes then were allowed to feed upon a healthy guinea pig (0) on the fourth day following their initial feeding and every second or third day thereafter, as long as mosquitoes survived. This pig developed posterior paralysis 17 days following its initial exposure to infective mosquitoes and was destroyed. A brain emulsion of guinea pig 0 was then inoculated into pig 287, which on the eighth day showed posterior paralysis, a temperature of 103.6° F. and a weight loss of 80 grams. This brain was in turn inoculated into three other pigs, each showing quite characteristic symptoms, weight loss, and high temperatures, and reaching the moribund state in from four to six days following the injection.

A portion of the brain emulsion of guinea pig 287 was also passed through a Berkefeld N filter; four pigs receiving this filtrate failed to succumb. Aerobic cultures and microscopic smears of this emulsion did not reveal evidence of bacterial contamination. The unfiltered emulsion continued to destroy guinea pigs for four successive passages, but at this point attempts were unsuccessful because the virus, if present, suddenly became inactive, either through faulty preservatives or from other unknown causes. Attempts to recover virus from older emulsions of this series were also unsuccessful. Since this case was not followed out with subsequent injection of horses, complete information is lacking in this instance. The preliminary findings, however, strongly suggest that equine encephalomyelitis virus was involved.

In addition to the three cases here discussed, there were also other instances where transmission of virus by mosquitoes was suspected, paralysis and other suggestive symptoms being in evidence. Subsequent inoculations with emulsions of such brains failed to produce conclusive evidence.

Our studies on the Utah insects as vectors of equine encephalomyelitis included some trials with simulids and horseflies as well as 86 trials with mosquitoes, in the majority of which *Aedes dorsalis* was used. The question may be asked: Why did such a large proportion of the transmission trials result in

failure? The writers consider that lack of information concerning how native marsh mosquitoes may be kept alive and vigorous for a sufficient time to allow virus multiplication within the mosquito to be one of the greatest handicaps. This was particularly true with reference to many of the earlier trials. Fifty additional attempts at transmission, using naturally infected horses for the initial feeding of mosquitoes, were all negative. It is likely that these horses were too far advanced in the course of the disease to show appreciable concentration of virus in the blood elements, even though mosquitoes often were fed upon horses a few hours after visible symptoms were first observed and while the temperature was as high as 105° F. It is not the contention of the writers that insect vectors are the only means of spreading equine encephalomyelitis. Some factors in connection with the epizoölogy, such as isolated winter cases, suggest other means of transmission. Work thus far reported does suggest, however, the possibility that blood-sucking insects do play a part in summer dissemination.

SUMMARY

In two different attempts, *Aedes nigromaculis* was found to transmit equine encephalomyelitis (western type) from affected guinea pigs to pigs not so affected.

Some attempts indicate that *Aedes dorsalis* may transmit the disease as did *A. nigromaculis*. Most attempts at transmission with *Aedes dorsalis* resulted in failure.

ACKNOWLEDGMENTS

The authors wish to acknowledge their indebtedness to J. A. Rowe, O. G. Larson and T. O. Thatcher, all of whom assisted in this investigation.

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Great Britain Breeding More Heavy Horses

The breeding of heavy horses, which has been declining steadily in Great Britain for the past twelve years, is showing signs of recovery, according to the *Scottish Farmer*. The number of foals is increasing, and the number of heavy stallions traveling in Great Britain in 1934 was the largest for ten years.

A NOTE ON THE NON-POISONOUS PROPERTIES OF OSAGE ORANGES (MACLURA POMIFERA)*

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Unidentified losses in animals on pastures where Osage oranges (*Maclura pomifera*) are available have, on occasions, prompted clinicians to conjecture as to the poisonous properties of this fruit for animals. Pammel's "Manual on Poisonous Plants" suggested the possibility of the leaves and fruit being more or less poisonous, although experimental evidence in support of these materials having poisoned animals has not come to our attention. In a personal communication, Dr. A. B. Clawson, plant physiologist, U. S. Department of Agriculture, suggests that published information would appear to indicate that Osage oranges are not likely to cause poisoning in farm animals. In support of this contention, he refers to the *Wallace Farmer*, 1915, Vol. 40, No. 49, p. 1584, in which a Kansas correspondent reports that his horses are fond of Osage oranges. One man had fed six or eight of the balls per horse daily without any injurious effects. Each of the horses in question is said to have eaten two or three bushels of the fruit during the fall. He states also that the Kentucky Experiment Station fed about six of the balls daily to each of their dairy cows for about three weeks without injurious effects.

In Champaign County, Illinois, October 13, 1934, two horses suffered from a fatal undiagnosed syndrome. These animals were in a pasture containing an abundant supply of Osage oranges. In a survey of the premises by Dr. A. E. Campbell, of Rantoul, an examination of the Osage oranges on the ground suggested that horses had possibly eaten some of the fruit.

The prevalence of Osage oranges in different sections of Illinois prompted orienting tests to note the effect of feeding the fruit

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and the expressed juice of the fruit. For this purpose one liter of the freshly expressed juice obtained from twenty Osage oranges was administered to a 1,400-pound Percheron mare, *via* stomach-tube. A Guernsey cow weighing approximatey 800 pounds was given a similar amount into the rumen, *via* stomach-tube. No ill effects were observed in these animals, as judged by clinical observation. Samples of the expressed juice were passed through Mandler filters. Notwithstanding that *Escherichia coli* was demonstrated bacteriologically in the filtered juice, injection of 1 cc to 4 cc subcutaneously into guinea pigs, rabbits and pigeons failed to suggest the toxic properties of the expressed juice.

Five chickens were fed eight Osage oranges that had been ground and mixed with mash. The mixture was fed continuously over a period of 17 days. The fowls were observed for a period of 17 days following the ingestion of fruit. No symptoms of illness were detected.

SUMMARY

1. The expressed juice of Osage oranges (*Maclura pomifera*), administered to a horse and a cow *via* mouth in one-liter doses, failed to induce clinical symptoms of poisoning.

2. The non-sterile, filtered juice injected subcutaneously into guinea pigs, rabbits and pigeons failed to produce toxic symptoms.

3. The feeding of Osage oranges disguised in mash to chickens failed to induce symptoms.

A CONGENITAL CAPILLARY HEMANGIOMA OF THE SKIN OF A THREE-DAY-OLD LAMB*

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Among the neoplasms affecting lower animals those consisting principally of blood-vessels which proliferate independently are not of common occurrence. In man, however, according to Mallory,[1] this type of growth is relatively common and is often of congenital origin.

Some of these vascular naevi have their anatomic inception in the subcutis and are ordinarily benign, merely constituting a blemish. They usually spread by infiltration of the surrounding

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tissues, although several growths may appear spontaneously suggesting primary multiple origin. Some cases, however, show apparent widespread metastasis, with tumors appearing in the different visceral organs.

Feldman,[2] in reviewing the literature on neoplasms of animals, found comparatively few hemangiomas reported. With one exception, the cases cited occurred in adult animals. In this case, congenital multiple endotheliomas were seen in the lungs of a one-day-old colt. In a series of 535 new growths obtained from lower animals, Feldman reports ten hemangiomas. They occurred

FIG. 1. One-half of tumor showing outer surface and incised surface.

as follows: four among 230 tumors obtained from cattle, three among 43 tumors of horses, two among 81 tumors of dogs and one among 41 tumors of sheep.

The subject in this case was a three-day-old, well-developed lamb, autopsied by Dr. W. T. Huffman, field veterinarian for the U. S. Bureau of Animal Industry in Utah, to determine the cause of death. In the course of the examination, he noted a bluish-black, raised growth on the ventral surface of the neck. The tumor was limited to the skin. Secondary growths were not ob-

served elsewhere in the carcass. Grossly, the tumor appeared melanotic. The lamb had apparently died of other causes.

The growth (fig. 1) is round, with a roughened, corrugated surface, and was attached to the skin by a rather broad base. It was 50 mm in diameter, 15 mm through the thickest part, and weighed approximately 55 grams. Numerous hair follicles were noted in the skin covering the body of the tumor.

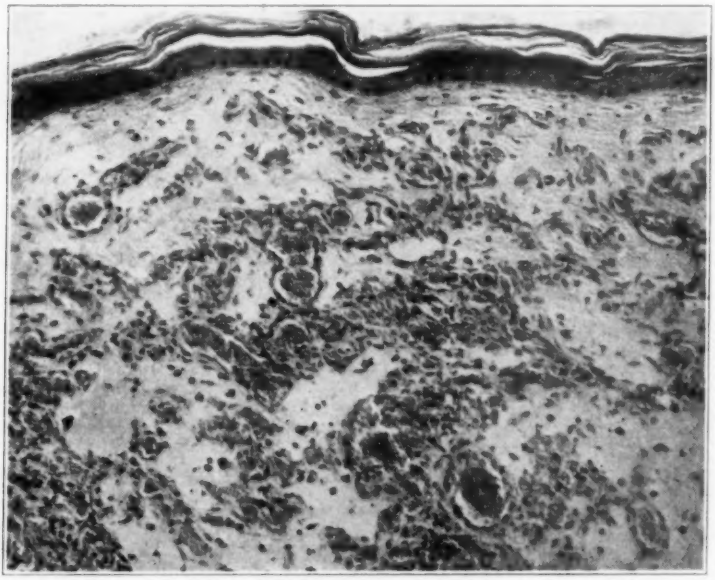

FIG. 2. Photomicrograph of tumor showing epithelial covering and a number of well-defined blood-channels filled with blood (x 150).

Histologically (fig. 2) the growth is composed of numerous blood-channels, varying in size and which are lined with a single layer of endothelial cells. The majority of the blood-channels appear to be well defined and many are filled with blood. The endothelial cells rest on a fine connective tissue stroma. Cross-sections of hair follicles are seen in the sections. The tumor is covered with epithelium, the peripheral layer showing keratinization. The color of the tumor is undoubtedly due to the excessive amount of blood in the vascular bed of the tumor.

Although many tumors of this type in man are often observed in the new-born, very few cases are reported occurring in new-born animals. Perhaps some of the hemangiomas observed in adult animals were of congenital origin, but because of their

clinical insignificance attracted no particular attention. The occurrence of a hemangioma in a three-day-old lamb lends to the belief of some veterinary pathologists that this type of tumor may be of congenital origin in lower animals.

ACKNOWLEDGMENTS

The writer is indebted to Dr. W. T. Huffman, Bureau veterinarian, stationed at Salt Lake City, Utah, for supplying the tissue for this report, and to Dr. W. H. Feldman, of the Mayo Foundation, Rochester, Minn., for preparing the photographs.

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DIABETES INSIPIDUS IN A BOVINE MALE*

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Diabetes insipidus has been defined by Dorland as "a chronic disease, marked by great thirst and the passage of a large amount of urine with no excess of sugar. It is often attended by voracious appetite, loss of strength, and emaciation." Diabetes insipidus must be distinguished from polyuria, which is a transient condition.

According to Hutyra and Marek,[1] spontaneous diabetes insipidus is infrequently seen among animals. It has occurred most frequently in horses. A horse so affected may drink as much as 100 liters of water daily, and void 40 to 60 liters of urine in 24 hours. The urine is water-like, does not have any odor, and has a very low specific gravity.

The causes of diabetes insipidus of animals have been variously ascribed to the ingestion of certain plants and foods, long-continued administration of diuretics, injuries to the liver and vertebral column, and infection. This condition has not been observed to occur as a complication of disease of the central nervous system. A possible sex gland relationship is suggested in the observations of Moiroud. He mentioned that stallions appear to be more disposed to diabetes insipidus than mares and geldings do.

The present knowledge of the cause of diabetes insipidus of man was recently reviewed by Futcher.[2] The chronological or-

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der of some of the important advances in the field, according to Futcher, would appear as follows: Thomas Willis, in 1674, was the first European to describe the sweet taste of urine and is credited with having differentiated the saccharine and non-saccharine varieties. However, Johnn Peter Frank, in 1794, was the first definitely to recognize that there are two distinct varieties of diabetes. Claude Bernard, in 1849, reported that puncture of the so-called diabetic center, which is situated in the floor of the fourth ventricle, caused polyuria with glycosuria in dogs. His results were confirmed by Echard and by Kahler, in 1870. In 1905, Erick Meyer advanced the theory that diabetes insipidus was the result of the inability of the kidneys to concentrate the urine. This theory, however, later was abandoned. Harvey Cushing, in 1910, reported his results with the removal of the posterior lobe of the pituitary body in dogs. He observed that a transient polyuria frequently followed this operation. Similar results have since been observed by other investigators, following experimental injury of the hypothalamus and the tuber cinereum. E. Frank, in 1912, reported the first clinical case which revealed a relationship between diabetes insipidus and lesions of the pituitary body. In 1913, Harvey Cushing, in his Shattuck lecture, mentioned that in his first 100 cases of disease of the pituitary body, six showed marked polyuria. Simonds, in 1913, reported a case of diabetes insipidus in which the patient, who was a woman, had a metastatic neoplasm in the posterior lobe of the pituitary body. Numerous similar cases have been reported since.

A further pituitary relationship was demonstrated in 1913, by von der Velden of Germany, and by Farini and Cecarroni of Italy,[3] who reported that subcutaneous injections of extracts that were made from the posterior lobe of the pituitary body caused prompt but temporary relief and control of excessive thirst and polyuria in patients affected with diabetes insipidus. Blumgart, in 1922,[4] reported that similar results were obtained when pituitary extract was applied intranasally in the form of a spray or on a swab. These results have since been confirmed by many other investigators.

Futcher mentions three views of the cause of diabetes insipidus, which have been advanced since 1910. The functions of the pituitary body and hypothalamus held a prominent place in each of these views.

Lower and Johnston, in 1931,[5] reported the results of their studies of the probable causes of hypertrophy of the prostate gland. They discussed the interrelationship of the sex glands

and the pituitary body. They said that Fichera, in 1905, observed that the pituitary body enlarges following castration and that similar observations were reported by Haitai, in 1913, by Smith and Engle, in 1927, and by Smith and MacDowell, in 1930. Lower[6] has since reported more experimental data which further tend to show a definite interrelationship of the sex glands and the pituitary body.

Because of the extensive clinical evidence and experimental data which tend to show a relationship of diabetes insipidus and pituitary function, and an interrelationship of the sex glands and the pituitary body, it is thought that the following case of diabetes insipidus in a bovine male merits being reported:

A one-year-old Holstein bull was observed to develop excessive thirst and polyuria rather suddenly. The cause of this could not be attributed to his diet or care, as several other male bovines in the same stable, which received similar food and care, remained normal. The symptoms could not be attributed to his habits or to any treatment which was administered. The symptoms became progressively more marked. After three weeks, it was estimated that he voided 40 to 50 liters of urine daily. An automatic drinking-cup furnished him an unlimited supply of water and he drank excessive quantities. Examination of the blood at this time revealed no significant changes. The urine, however, was odorless, colorless, sugar-free and of very low specific gravity. Indeed, it was little more than water. This animal had a voracious appetite, though he manifested progressive loss of flesh.

Soon after the onset of this disease an attempt was made to control the polyuria by reduction of his water intake. This appeared merely to aggravate his symptoms and was discontinued after two days. When it appeared certain that this was not a transient polyuria, orchectomy was done at the request of the owner, who was a physician. The result of this operation was most striking. Marked improvement was noted after 24 hours, and within three days all symptoms of excessive thirst and polyuria had vanished.

The prompt and specific effect of castration in this case of polyuria in a bovine male tends to support the theory of a relationship of the pituitary body and sex glands with diabetes insipidus.

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Fighting Animal Diseases in Ohio

Ohio newspapers have been generous with their space in publicizing the federal and state programs for the eradication of bovine tuberculosis and Bang's disease. The Sunday feature section of the *Columbus Dispatch* for April 14 carried a half-page illustrated article devoted to an intelligent discussion of the campaigns against these diseases that are under way in Ohio under the supervision of Dr. A. J. DeFosset, of the U. S. Bureau of Animal Industry. Engaged in the work are 80 veterinarians, 12 laboratory technicians and an office staff of 14 clerks.

The article points out that the annual human death-rate from tuberculosis in Ohio has dropped from 145 per 100,000 population in 1918 to 55.1 in 1932, and credits the campaign against bovine tuberculosis for its fair share in this reduction. Today Ohio is an accredited state despite the fact that only ten years ago tuberculosis among dairy cattle in some townships in the northeastern part of the state was as high as 76 per cent, according to figures quoted by Dr. DeFosset in the article. A strenuous fight is also being waged against Bang's disease. More than 20,000 cattle afflicted with the disease have been condemned during the past six months.

Chimpanzee at London Zoo Has Blessed Event

A bronze medal was recently awarded to Mr. Frank Shelley, head keeper of the apes and monkeys in the London Zoölogical Gardens, for his success with the birth of the first chimpanzee to be bred and born in the Gardens. The Zoölogical Society presented the medal. A money award also was made to Mr. Shelley and to Mr. A. S. Budd, his assistant. According to the *Veterinary Record*, the two keepers had won the complete confidence of the chimpanzee, and attended it day and night while the birth was expected and for some days afterward.

Is not true leisure one with true toil?—DWIGHT.

THE INTERNAL PARASITES OF DOMESTIC ANIMALS. Thomas W. M. Cameron, Research Professor of Parasitology, McGill University, Director of the Institute of Parasitology, Macdonald College, Canada. 292 pages and 137 illustrations. A. and C. Black, Ltd., London; The Macmillan Co., New York. 1934. Cloth, $4.

Two books on the subject of parasitology of domesticated animals were published in English during 1934, the first by Dr. H. O. Mönnig* and the second by Dr. Cameron. The book by Mönnig deals with the diseases caused by worms, insects, mites and ticks, whereas Cameron includes the protozoan and worm parasites.

The latter book is well written and well arranged and the nomenclature appears to be valid. The text is rather brief, for example, only thirty pages are devoted to a discussion of protozoan parasites. However, as the author explains, the book is written primarily as a guide, to be supplemented by more detailed study of the subject in the classroom and laboratory. Furthermore, leading references are cited that will point the way toward a more detailed study of individual parasites or of groups.

The time has passed when a single author can hope to include in one volume (to be sold at a reasonable price) all the information necessary to adequately answer questions in the ever-widening field of veterinary parasitology. Hence, if those interested wish to obtain authoritative information, they must consult various books, monographs, bulletins and periodicals from time to time and take from them the facts that are needed.

A refreshing feature of this book is the originality of the photographs and drawings, although in some of the life-cycle diagrams, improvement might be made by using simply the host name in place of a drawing.

Exception might be taken to the statement that flotation technics for the detection of helminth ova and coccidia have been greatly overvalued as a routine diagnostic measure for the practitioner. In this country, at least, there is a tendency to increase

*Jour. A. V. M. A., lxxxv (1934), n. s. 38 (4), pp. 546-547.

the use of such technics, particularly by the small-animal practitioner.

As a whole, however, Dr. Cameron's book can be highly recommended and its use should be encouraged in a profession whose reference needs are great but inadequately supplied.

E. A. B.

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Serologic Relationship of Brucella and Pasteurella. L. E. Starr and G. E. Snider. Reprint from *Jour. Inf. Dis.*, lv (1934), pp. 384-389.

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Erythrocyte Production in the Bone Marrow of the Pigeon. H. E. Jordan and E. P. Johnson. Reprint from *Amer. Jour. Anat.*, lvi (1935), 1, pp. 71-95. Illus.

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Zoo Men See Solution for Their Problems

The issue of *Parks & Recreation* for March, 1935, contains the first part of the paper by Dr. J. A. Campbell, of Toronto, Ont., which was published in the May, 1934, JOURNAL of the A. V. M. A., under the title of "Some Observations Relative to Ailments of Inmates in a Zoölogical Collection." In a foreword to the article, the editor of the magazine points out that "the article contains much material of paramount interest to zoo men and indicates a solution for many of the problems which continually appear in zoölogical parks." The reprinted article will be continued through two subsequent issues of the magazine.

COMPLEMENT-FIXING ANTIBODIES IN CHICKEN LEUKOSIS OR ERYTHROBLASTOSIS. O. Thomsen, J. Engelbreth-Holm and A. Rothe Meyer. Abst. Arch. Path., xix (1935), 1, p. 121.

About 20 per cent of the serums of chickens with experimental leukosis or erythroblastosis gave positive complement-fixation with an antigen prepared from the immature blood cells. That the reaction was due to a development of isoantibodies against the injected blood-cells and not to antibodies, and not to antibodies against the introduced virus became apparent from the following observations: (1) The serums of chickens which were inoculated with blood cells of other normal chickens fixed complement quite as well as the serums of infected chickens. (2) The complement-fixing antibodies in the serums of chickens which were inoculated with leukosis and of those which were treated with normal blood-cells were readily absorbed with normal red blood-cells. Inoculation of chickens with red blood-cells of other normal chickens did not increase their resistance against subsequent inoculation with the virus of leukosis.

EFFECT OF RESECTION OF LARGE FRACTIONS OF RENAL SUBSTANCE: AN EXPERIMENTAL STUDY. Raymond B. Allen, Jesse L. Bollman and Frank C. Mann. Arch. Path., xix (1935), 2, p. 174.

A series of eight dogs, aged from five to six months, were subjected to resection of large fractions of renal substance; the largest fraction was not less than three-fourths. After an interval of several months, the degree and nature of the hypertrophic response of the remaining renal tissue were studied. Transitory increases occurred in the concentration of the blood urea following resection, but as little as one-fourth of the renal substance was capable of maintaining a normal concentration. Readings of the blood pressure made by indirect and direct methods were normal. In one animal, 72.8 per cent of the renal substance was removed at one operation and the animal survived. In another animal, somewhat more than three-fourths of the

renal substance was removed in two stages by waiting for compensatory hypertrophy to occur in the interval and the animal survived. The larger the proportion of total renal substance resected the greater is the relative hypertrophy which occurs, both in terms of mass of kidney and in terms of the size of the renal corpuscles and tubules. The observations suggest that compensatory renal hypertrophy is a physiologic reaction.

THE DESTRUCTIVE ACTION OF BLOOD-SERUM ON HEMOGLOBIN. L. Doljanski and O. Koch. Abst. Arch. Path., xix (1935), 1, p. 111.

When blood-serum was added to a freshly prepared solution of hemoglobin in Tyrode's fluid and the mixture incubated, the formation of methemoglobin and later hematin was noted. Similar changes occur in solutions of hemoglobin not exposed to the action of serum, but in the presence of the latter the process is more rapid and more marked. Human serum had a more pronounced action in this respect than chicken or rabbit serum. The authors interpret their findings as evidence of a toxic or destructive action of normal blood-serum on hemoglobin. A protective mechanism that prevents action of the serum on the hemoglobin of the corpuscles under normal conditions is postulated.

THE AMERICAN DOG TICK, DERMACENTOR VARIABILIS, AS A HOST OF BACTERIUM TULARENSE. C. B. Philip and W. L. Jellison. Abst. Arch. Path., xix (1935), 2, p. 258.

American dog ticks, *Dermacentor variabilis*, in the adult stage and in the larval stage, were experimentally infected with *Bacterium tularense*. Larvae from the ticks in the adult stage fatally infected a white-footed mouse. Resultant engorging nymphs were shown to contain the organism in virulent form, which in some instances apparently caused the death of the ticks *in situ;* however, demonstrable infection was not produced in some of the host animals. Further evidence of generation to generation continuity of *B. tularense* in these ticks was secured by the injection of partial batches of eggs from two additional infected ticks. Nymphs reared from infected larvae produced fatal infections in two guinea pigs. Infection was prolonged by resultant adults in separate guinea pigs both by feeding and by injection. Tests with this and other species of ticks suggested that *B. tularense* is not entirely adapted to continued residence in ticks through their developmental cycle, since the ticks them-

selves sometimes die while still attached to the host animal and occasionally without infecting the host. Since the continuity of infection in the tick has been demonstrated, *D. variabilis* must be kept in mind as a possible source of human infection.

ELIMINATION AND CROSS-INFECTION EXPERIMENTS WITH TRICHOMONADS FROM FOWLS, RATS AND MAN. Robert Hegner and Lydia Eskridge. Amer. Jour. Hyg., xxi (1935), 1, p. 135.

Trichmonads from fowls, rats and man were used and chicks and rats were employed as experimental hosts. Viscous substances including agar-agar and gum arabic, when added to the food of rats infected with rat trichomonads, brought about a pronounced decrease in the number of these flagellates present but did not eliminate them entirely. Chicks infected with fowl trichomonads showed no decrease in the number of these flagellates when psyllium or flax seed was added to the diet. It was demonstrated that 1 per cent copper sulphate is destructive to trichomonads. Carbarsone in 1 per cent concentration in 1 per cent sodium bicarbonate solution, given to rats in place of water, was effective in about five days in eliminating all of the trichomonads from the experiment animals. Other intestinal protozoa, amoebae, giardias, Hexamitus and Chilomastix did not seem to be affected by this latter treatment. Carbarsone given to chicks with cecal infections of fowl trichomonads eliminated the flagellates in from one to seven days; it was possible to reinfect the chicks when treatment was discontinued.

ARTIFICIAL IMMUNIZATION OF RATS AGAINST TRICHINELLA SPIRALIS. O. R. McCoy. Amer. Jour. Hyg., xxi (1935), 1, p. 200.

The majority of rats given six intraperitoneal injections, at five-day intervals, of living trichina larvae, heat-killed larvae, or dried and powdered larvae developed some degree of immunity against a subsequent light infection with *Trichinella* spiralis. The degree of immunity in the individual animals varied from none to practically complete. The injection of living larvae was usually more effective in establishing immunity than the injection of either heat-killed larvae or dried and powdered larvae. Artificially immunized rats showed little or no resistance to the initial development of adult worms in the intestine, but the worms were lost more rapidly than in control animals. This of course would result in a smaller amount of muscle invasion in the

immunized rats. The immune state produced by the injections is apparently of the same general nature as that brought about by actual infection; in each instance the mechanism is directed against the intestinal stages of the parasite. The former immunity is not nearly so potent as the latter and is much more easily broken down by large doses of larvae.

THE STABILITY OF THE COLONY MORPHOLOGY AND PATHOGENICITY OF BCG. Dorothy M. Behner. Amer. Rev. Tuber., xxxi (1935), 2, p. 174.

Variation in colony morphology under different environmental conditions was not accompanied by enhanced pathogenicity. *BCG* did not acquire virulence in laboratory culture on liquid media containing either normal human serum or homologous antiserum or in the depths of nutrient broth or by successive selection for propagation of smooth-appearing colonies on solid media or by repeated animal passage. A virulent, bovine-type strain was found to be resistant to laboratory modification but some attenuation in virulence was produced together with a slight change in colony morphology with the appearance of some characteristics usually associated with the rough variant. Avian strains were found to be labile, developing changes in colony morphology in both smooth to rough and rough to smooth directions, and with also some marked variations of each type together with accompanying changes in pathogenicity under environmental conditions similar to those imposed in *BCG* strains.

OBSERVATIONS ON MIXTURES OF CASEIN AND NATIVE OX SERUM ALBUMIN. Mavis Freeman. Austral. Jour. Exp. Biol. & Med. Sci., xii (1934), 3, p. 155.

There is some interaction of casein and ox serum albumin when the former is precipitated in the presence of the latter. It is suggested that a small fraction of casein, relatively low in phosphorus content, tends to associate with the serum albumin. A small fraction of casein of high phosphorus content dissolves when casein is extracted alone with 1 per cent sodium chloride solution after precipitation with acid. The more of this protein that is extracted the more alkaline the pH of the precipitation. This fraction soluble in 1 per cent sodium chloride has a lower phosphorus content than casein. The production of this fraction may be caused by a mixture of the two proteins or by a splitting of the original casein.

THE NEMATODES OF SHEEP IN MANAWATU DISTRICT, NEW ZEALAND. J. H. Tetley. Jour. Helminth., xii (1934), 4, p. 123.

Twenty-five species of nematodes were found to be present in sheep in Manawatu sheep, the species of greatest economic importance being in most cases those of importance in other temperate climates. Most species of nematode infection are possible throughout the year; infective stages are able to survive the extremes of summer and winter. The prominence of the fat-lamb industry means the raising of a population susceptible to worm infection. Cattle are the chief reservoir host of nematodes of sheep in this district. This occurs principally where dairying is combined with sheep farming in respect to some species of worms (*Cooperia oncophora, C. punctata*, and *Ostertagia ostertagi.*)

FURTHER OBSERVATIONS OF THE EFFECT OF HEAVY STOCKING ON THE WORM BURDEN UNDER A SYSTEM OF ROTATIONAL GRAZING. D. O. Morgan and J. N. Oldham. Jour. Helminth., xii (1934), 4, p. 177.

There is a surprising difference in the relative frequency of the species of worms present when sheep are grazing on the same plot. In three experiment sheep the predominating species differed in each animal. *Chabertia ovina* and the smaller trichostrongyles were the commonest species picked up during the winter months. Larvae in all stages of development were observed during the winter months along with adults of the species: *Chabertia ovina, Trichostrongylus axei, Ostertagia circumcincta* and *O. trifurcata. Bunostomum trigonocephalum* also seems to fall within this group. Summer months appear to be more suitable for the development of *H. contortus.* Some sheep apparently develop an immunity to this species. The authors conclude that for older sheep the autumn and winter months are the most critical to stock on heavily contaminated pastures.

SOME POINTS IN THE EARLY DEVELOPMENT OF CYSTICERCUS PISIFORMIS (Bloch 1780). S. G. Solomon. Jour. Helminth., xii (1934), 4, p. 197.

Onchospheres, after being swallowed by the rabbit, migrate from the stomach—not from the intestines—into the portal vessels from which they are carried to the liver. There are no hooks or penetrating apparatus; the end which later forms the scolex is densely granular while the opposite pole, which will

form the bladder, is hyaline. They wander through the liver and, by the end of the second week, begin to migrate from the liver into the body cavity. Encystment now takes place. In refractory subjects encystment may take place in the liver.

A STUDY OF THE GOLGI APPARATUS OF THE ADRENAL GLAND. Goeffrey Bourne. Austral. Jour. Exp. Biol. & Med. Sci., xii (1934), 3, p. 123.

Two distinct types of Golgi apparatus have been found in the adrenal cortical cells, the compact and the diffuse. The latter is regarded as the hypertrophied form of the compact type. The cells of the *zona reticularis* are shown to possess a Golgi apparatus which takes the form of either a mass of granules or a condensed black knot closely associated with the nucleus. This fact is in agreement with the view that the *zona reticularis* is a zone of degenerating cells. It is concluded that the Golgi apparatus and vitamin C are jointly concerned in the production of lipoid material, which may be cortical hormone, in the cells of the adrenal cortex.

Fifty Years in General Practice

Dr. Thomas E. Jones, of Newark, Ohio, who entered upon his 51st year of general practice in Newark on April 1, 1935, was the subject of a three-column biographical sketch in the *Newark Leader* for March 28.

Graduating from the Ontario Veterinary College in 1885, Dr. Jones located in Newark at the time that Grover Cleveland was beginning his first term as President of the United States. He has seen many changes in the past half-century, including changes in modes of travel. He has answered calls by walking, horseback, buggy, hack, street car, automobile and train. He had not yet used an airplane, but he may do so before he takes down his shingle.

Dr. Jones was born in Wales, January 12, 1861. When he was five years old, the family emigrated to America and joined the settlement of Welsh pioneers in and near Granville, Ohio. He first attended Denison University, at Granville, and then entered the Ontario Veterinary College. At 74, he is keen and alert, and finds his professional duties as absorbing as when he entered upon his career. Dr. Jones is a member of the A. V. M. A., having joined in 1920.

Regular Army

Captain Herbert M. Cox is relieved from duty at Plattsburg Barracks, N. Y., effective at such time as will enable him to proceed to New York, N. Y., and sail on the transport scheduled to leave that port on or about June 6, 1935, for the Panama Canal Department, for assignment to duty with the Medical Department.

Major Philip H. Riedel is assigned to duty at Fort Sam Houston, Tex., upon completion of his present tour of foreign service in the Panama Canal Department.

Each of the following named officers of the Veterinary Corps is relieved from further assignment and duty at the station as specified after his name, effective in time to comply with this order, and will proceed to Washington, D. C., and report to the commanding general, Army Medical Center, on or about August 28, 1935, for duty for the purpose of pursuing a course of instruction at the Army Veterinary School:

Lt. Col. Isaac O. Gladish, Fort Bragg, N. C.
Major Kenneth E. Buffin, Fort Sam Houston, Tex.
Major Fred W. Shinn, Fort McIntosh, Tex.

Lt. Col. Daniel B. Leininger is relieved from further assignment and duty at the Presidio of Monterey, Calif., effective in time to proceed to San Francisco, Calif., and sail on the transport scheduled to leave that port for New York, N. Y., on or about July 30, 1935; upon arrival in New York, will proceed to Washington, D. C., and report to the commanding general, Army Medical Center, for duty for the purpose of pursuing a course of instruction at the Army Veterinary School.

Lt. Col. Jean R. Underwood is relieved from further assignment and duty at Fort Bliss, Tex., effective in time to proceed to San Francisco, Calif., and sail on the transport scheduled to leave that port for New York, N. Y., on or about April 20, 1935; upon arrival in New York, will proceed to Washington, D. C., and report to the commanding general, Army Medical Center, for duty.

Major Oness H. Dixon, Jr., is relieved from assignment and duty with the Chicago quartermaster depot, Chicago, Ill., effective at such time as will enable him to comply with this order, and will report not later than August 20, 1935, to the commandant, the Quartermaster Corps Subsistence School, Chicago, Ill., for duty as a student in the 1935-1936 course.

Major George L. Caldwell is relieved from duty at Fort Riley, Kan., effective at such time as will enable him to proceed to Chicago, Ill., and report not later than August 20, 1935, to the commandant, the Quartermaster Corps Subsistence School, for duty as a student in the 1935-1936 course.

Captain John L. Owens is relieved from further assignment and duty as student, the Quartermaster Corps Subsistence School, Chicago, Ill., effective upon completion of his present course of instruction, and will then report to the commandant, that school, for duty as instructor.

Major Sherman R. Ingram, having been examined for promotion by a board of officers and found physically disqualified for the duties of a lieutenant colonel of the Veterinary Corps, by reason of disability incident to the service, his retirement by the President from active service as a lieutenant colonel under the provisions of the act of Congress approved April 23, 1908, and the act of Congress approved April 23, 1930, is announced to become effective March 31, 1935, with rank from March 1, 1935, the date upon which he would have been promoted to that grade by reason of length of commissioned service if found qualified.

Notice of Examination for Appointment in the Veterinary Corps, Regular Army

An examination of applicants for appointment as second lieutenants, Veterinary Corps, Regular Army, under the provisions of AR 605-20, will be held within the continental limits of the United States, from July 8 to 13, inclusive.

Application blanks, Form No. 62, A. G. O., may be obtained from the Adjutant General or the Surgeon General, U. S. Army, Washington, D. C., or from the Commanding Officer or Surgeon of any military post or station, and when completed should be forwarded direct to The Adjutant General, U. S. Army, Washington, D. C.

The applicant must be a male citizen of the United States, between the ages of 21 and 29-9/12 years, and a graduate of a recognized veterinary college.

Applications received after June 25, 1935, will not be considered.

Veterinary Reserve Corps

New Acceptances

Cleaver, Fred Everett...Capt....20 E. Main St., Avon, N. Y.
Lee, Chester Daniel.....1st Lt...1120 Harding Ave., Ames, Iowa.
Ludloff, Otto Walter....2nd Lt..1321 Palolo Ave., Honolulu, T. H.
Foote, Bernard Eugene..2nd Lt..3-4 State War Memorial Bldg., Nashville, Tenn.
Mims, Jos. Bryan.....2nd Lt..500 W. Henderson St., Cleburne, Tex.

Active Duty for Reserve Corps Veterinary Officers

The War Department contemplates calling to active duty, with their own consent, for periods of six months or longer, one hundred or more Veterinary Reserve officers in the grades of second lieutenant, first lieutenant and captain, for service with the Civilian Conservation Corps. These veterinarians must be qualified in meat and dairy inspection.

An Abrupt End to a Long Life

A life of dignity and service such as it is given few Rhode Island Red hens to enjoy was ended abruptly at the age of 22, when the automobile of Dr. G. S. Hicks (Geo. Wash. '12), of Milford, Mich., accidentally ran down and killed his prized possession. The hen had been in the flocks of Dr. Hicks during the entire 22 years of her lifetime.

More Honors for Dean Francis

Dean Mark Francis, of the School of Veterinary Medicine, Texas A. and M. College, was honored during the Southwestern Exposition and Fat Stock Show, at Fort Worth, Tex., March 16-24, 1935. He was invited as the guest of the Exposition and the Texas Hereford Breeders' Association, and was paid especial tribute by the Breeders' Association for his contributions to the live stock industry in Texas, at a "Mark Francis Dinner" on March 19, the date of his 72nd birthday anniversary. More than 350 guests were present to honor Dr. Francis.

When he returned to the College, Dean Francis was honored by the student chapter of the American Veterinary Medical Association at a second birthday dinner, on March 25. All the deans of the Texas A. and M. College and other faculty members spoke in tribute to Dean Francis, and the speeches were broadcast over WTAW, the College broadcasting station. An unusual feature of the program was that three of the speakers, all prominent officials of the College, at one time studied under Dr. Francis. These included Dean E. J. Kyle, of the School of Agriculture, Dean C. H. Winkler, of the School of Vocational Education, and Dr. H. Schmidt, acting chief of the Division of Veterinary Science, Texas Agricultural Experiment Station.

Dean F. C. Bolton, of the School of Engineering, read a tribute prepared by President T. O. Walton, of the College, who had been called away on business. Dr. Walton praised Dean Francis' scientific achievements, chief of which was his pioneer work in trying to develop a method of immunizing cattle against Texas fever. He praised also the work of veterinary scientists in general.

Dean Francis, in a short talk closing the program, praised the coöperation, the team work, and the intelligence of his associates in carrying out the work of the College. "All I hope," he said, "is that I have the strength and intelligence to do my part."

TEXAS A. & M. COLLEGE STUDENT CHAPTER OF THE A. V. M. A.

J. O. Whitehead, of Smith Point, Tex., president of the student chapter, was chairman of the banquet committee, and Charles Coleman, of Alpine, Tex., a junior in the School of Veterinary Medicine, acted as toastmaster.

Pennsylvania Veterinary School to Celebrate

The School of Veterinary Medicine of the University of Pennsylvania will celebrate the fiftieth anniversary of its establishment, May 17-18, 1935. The occasion was to have been celebrated last year, but was postponed because of the Twelfth International Veterinary Congress and for other reasons. An interesting two-day program has been planned, and a letter has been sent to all alumni urging them to be present for the unusual event.

The program will be opened with the presentation of the Pearson Memorial, a bronze bust of the late Dr. Leonard Pearson ('90), dean of the School from 1897 to 1909. Dr. G. A. Dick ('04), the present dean of the School, will preside. The presentation address will be made by Dr. T. E. Munce ('04). Following the unveiling of the Memorial, it will be accepted on behalf of the School by Dr. Thomas S. Gates, president of the University of Pennsylvania. A feature of the first-day program will be the conferring of honorary degrees. President Gates will confer the degree of Doctor of Science on Drs. Louis A. Klein ('97) and C. J. Marshall ('94), of the University of Pennsylvania, and Dr. L. Van Es, of the University of Nebraska; and the degree of Doctor of Laws on Mr. J. Bertram Lippincott, president and treasurer of the Board of Managers of the Veterinary Hospital. Dr. Klein joined the faculty in 1909 as professor of veterinary hygiene and pathology, and succeeded Dr. Pearson as dean of the School following his death. He continued to serve as dean until 1930, when he resigned to devote his entire time to teaching and research. Dr. Marshall, who occupies the position of professor of veterinary medicine, has been connected with the School practically since his graduation. It is fitting that Mr. Lippincott should be honored, since it was the generosity of his family that made possible the founding of the School of Veterinary Medicine.

The plans for the afternoon include clinics in which students will participate; a program on milk hygiene, which will include visits to a number of dairy farms and a conference in Pearson Hall; and a visit to the Botanical Gardens and the University Museum for the ladies. In the evening, a banquet will be held at the Hotel Pennsylvania.

On the second day, the annual meeting of the Alumni Society will be held, with Dr. Hiram P. Eves, of Wilmington, Del., presiding. Dr. Eves was a member of the first graduating class, in 1887. At the meeting, the Committee on History will make a report. It is expected that the history of the School, now in preparation, will be ready for distribution at this time. At 12:30 p. m., a buffet luncheon will be served in Pearson Hall, following which the alumni and their ladies will attend the Princeton-Pennsylvania baseball game. The procession to Franklin Field will be led by the American Legion Boys' Drum and Bugle Corps.

Catering to New York's Dogdom

Dogs owned by New Yorkers should have no internal worries at all, since the advent of the Canine Catering Company and the K-9 Kitchen. Both of these institutions promise the best of dietary guidance to Manhattan dogs and, for a nominal weekly payment, will deliver balanced rations that are guaranteed to satisfy the most finicky dog. According to the menu sent out by the Canine Catering Company, special diets for invalid or growing puppies will be prepared according to the advice of the veterinarian in attendance. Meals that have been "prescribed by the veterinary profession," are priced at 19 cents a pound and 11 cents a half-pound. A kennel meal (raw) is offered for 18 cents a pound and 10 cents a half-pound. "The K-9 Kitchen furnishes a perfectly balanced ration highly recommended by Dr. C. G. Rohrer, 40 W. 61st St., one of New York's most prominent veterinarians," according to its announcement. The Kitchen will supply a six- to eight-ounce daily ration for dogs of small and medium size, for $1 weekly. For sporting and medium large dogs, the cost for a 12-ounce to one-pound daily ration is $1.30 to $1.50 weekly. For large dogs the price is somewhat more. Meals from the K-9 Kitchen are delivered three times a week, in a sanitary container, an arrangement that saves the dog-owner the necessity of marketing and the trouble of cooking. After the dog has eaten, there remains the matter of his daily exercise. This is supplied by the Daily Dog Walking Service, Inc., which employs college boys and unemployed men to take the dog for short or long walks, as the owner wishes, the cost of the service being based on the amount of time subscribed for, on a daily or weekly basis.

He is well paid that is well satisfied.—SHAKESPEARE.

Doctor Smillie Made Member of Health Board

Dr. E. W. Smillie (Iowa '16), superintendent of the Department of Animal and Plant Pathology, Rockefeller Institute for Medical Research, Princeton, N. J., is the new veterinary member of the New Jersey State Department of Health, his appointment having been confirmed by the State Senate on March 19, 1935. Dr. Smillie takes the place of Dr. H. E. Winter (U. P. '10), of Plainfield, N. J., whose term expired last year.

DR. E. W. SMILLIE

New Jersey requires that one member of the Department of Health shall be a veterinarian, and, as far as is known, is the only state having such a law. The New Jersey law reads as follows:

> The Department of Health is hereby established, and the same shall be governed by a board of eleven members, to be known as the Department of Health of the State of New Jersey. Not more than six of the members of the board shall be members of the same political party, and all of said members shall be residents of this State, and two of the members of said board shall be women. At least three of the members shall be physicians, at least one a veterinarian, at least one a dentist, and at least two sanitary engineers.

Dr. Smillie entered the Department of Animal Pathology of the Rockefeller Institute as a research worker, July 1, 1916, and was promoted to his present position in 1919. He joined the A. V. M. A. in 1916, and served as Resident Secretary for New Jersey, 1923-1925. He is active in community affairs in his home town of Plainsboro and was secretary of the Plainsboro Council,

1923-1930. He is also a member of the Board of Trustees of Princeton Hospital, a Mason, and a member of Phi Gamma Delta, Phi Kappa Phi and Sigma Xi fraternities.

J. G. H.

Live Stock Loss Prevention Campaign Under Way

The program of the National Live Stock Loss Prevention Board, which was outlined in the December, 1934, issue of the JOURNAL, was given unanimous approval by the Executive Committee at a meeting in Chicago recently. The program includes an aggressive campaign of education designed to prevent the loss of $30,000,000 annually in bruised and injured live stock, the result of improper handling on the farms, in transit and at the stock yards. A committee is already at work on a statistical survey of the live stock loss situation.

Field activities of the Board are under the management of H. R. Smith, Live Stock Commissioner for the National Live Stock Exchange. Mr. Smith is assisted by four regional managers: Harry J. Boyts, of Sioux City, Iowa; Ray L. Cuff, of Kansas City, Mo.; Dr. W. T. Spencer, of Omaha, Neb., and W. A. Peck, of Saint Paul, Minn.

The officers and directors of the Board are representative of the variety of organizations concerned with live stock loss prevention. Officers serving their first terms are: Chairman, Earle G. Reed, of Columbus, Ohio, who represents the New York Central lines; vice-chairman, H. R. Davison, of Chicago, Ill., representing the Institute of American Meat Packers; secretary, Dr. H. Preston Hoskins, of Chicago, Ill., acting on behalf of the American Veterinary Medical Association; treasurer, C. H. McNie, of Chicago, Ill., acting for the Chicago and North Western Railroad.

In addition to the officers, the directors include: Fred S. Brooks, of the Rock Island lines; B. B. Brumley, of the Ohio Live Stock Coöperative Association; George W. Davies, National Association of Swine Records; E. P. Hoppel, Northern Pacific Railway Company; F. E. Knutzen, Swift and Company; L. W. Kube, Saint Paul Union Stock Yards Company; J. A. McNaughton, Los Angeles Union Stock Yards Company; F. R. Marshall, National Wool Growers Association; F. E. Mollin, American National Live Stock Association; Charles H. Schultz, Chicago Live Stock Exchange; F. M. Sherwood, Armour and Company; J. E. Thoman, Hartford Fire Insurance Company; William H. Weeks, Kansas City Stock Yards Company; P. O. Wilson, National Live Stock Marketing Association.

Members of the Board feel that now is the time to launch a well-planned program of live stock conservation, since the country is faced with reduced supplies and each individual meat animal is relatively more valuable.

Veterinarian Has Time for Civic Affairs

Dr. O. H. Person, mayor of Wahoo, Neb., had the honor of serving as president of the Nebraska League of Municipalities during the past year, the only veterinarian to hold such a position. That he filled the office with honor and distinction is shown by the progress made by the League, as reported by Nebraska

DR. O. H. PERSON

newspapers. The League is composed of 216 Nebraska towns and cities, 55 of which were admitted during President Person's term of office.

Dr. Person is now serving his eighth year, or fourth term, as mayor of Wahoo. He has been engaged in general practice in Wahoo since his graduation from the Kansas City Veterinary College in 1913.

A bear cub, a hybrid between the polar bear and the Kodiak bear, was born in the National Zoo, Washington, D. C., recently, and is believed to be the first instance of such a cross.

Time ripens all things. No man is born wise.—CERVANTES.

Live Stock Situation

Rapid and significant changes have taken place in the live stock industry since the beginning of 1934, according to Gerald B. Thorne, of the Agricultural Adjustment Administration, who gave facts about and figures on these changes at the 23rd annual Feeders Day meeting, College of Agriculture, Lincoln, Neb., April 18, 1935.

At the beginning of 1934, the cattle population of this country was 68,290,000. A year later, this number had declined to 60,667,000, a net reduction during the year of 11 per cent. The hog population declined even more sharply. From 52,212,000 head of swine on hand the first of last year, the number dropped to 37,007,000 by the last of the year, a decrease of approximately 35 per cent. The number of sheep decreased by 2,446,000 head, or about 5 per cent. The total on January 1, 1935, was 49,766,000 head. In spite of the drouth liquidation, however, sheep numbers still are above the 1929 level and also above the average of the past ten years.

The big reduction in cattle numbers was largely the result of reduction of feed caused by drouth. Government buying was inaugurated to relieve cattle producers and to conserve the meat of animals that otherwise would have died on the range. This action made it possible for farmers to keep somewhat larger numbers of animals on hand than they could have if they had not been relieved of distress stock. Altogether the government bought 8,296,398 head of cattle at a total cost of $111,778,192. In addition to these relief purchases, an unusually large number of cattle were disposed of through regular channels. Commercial marketings of cattle for 1934 were about 15 per cent larger than for the preceding year, according to Mr. Thorne. The greatest reduction of any one class of cattle in 1934 was in steers, which totaled about 20 per cent. Milk cows showed the smallest reduction, amounting to only 4 per cent.

Because of the sharp drop in the spring pig farrowings, the greatest reduction in live stock slaughter supplies has been in hogs. Federally inspected slaughter of hogs during the first three months of 1935 amounted to only 7,600,000 head, or 36 per cent under the number slaughtered during the corresponding period a year earlier. By comparison, commercial slaughter of cattle for the first quarter of 1935 was 10 per cent smaller than the first quarter of 1934, but sheep and lamb slaughter was slightly larger.

A consequence of wide general interest resulting from the material change in the total market supplies of live stock during recent months has been the advance in live stock and meat prices. The farm price of hogs in Nebraska advanced from $4.90 per hundred pounds on December 15, 1934, to $8.50 on March 15, 1935. The price of cattle advanced from $4.80 per hundred pounds to $8.30. Lambs advanced from $5.60 to $7.40 per hundredweight.

Mr. Thorne pointed out that, although live stock and meat prices during recent months have advanced much more than prices of most other products, they are not abnormally out of line. Consumers lose sight of the fact that, during the past three or four years, they have been able to purchase meat at bargain prices. Naturally, the recent rise in live stock and meat values—even though not out of line with other costs—seems exorbitant to the average consumer.

The Institute of American Meat Packers reports that the consumption of meat for the whole year of 1934 was 20 billion pounds as compared with the consumption of 18 billion pounds in 1933. The 1934 per capita consumption of meat was greater than in any year since 1907.

Cycles of overproduction and underproduction in the cattle and hog industry have been major hazards for many years, Mr. Thorne emphasized. The cycle represents a ruthless and wasteful system of keeping a working relationship over a period of years between supply and demand. Both its upswings and downswings usually overshoot the mark. The present is an opportune time for live stock producers to consider ways and means of controlling cycles of over- and underproduction. The important thing now simply is to prevent the increase in production from going beyond desirable limits.

Michigan-Ohio Veterinarians to Hold Clinic

Dr. E. C. W. Schubel, of Blissfield, Mich., secretary of the Michgan-Ohio Veterinary Medical Association, is planning a real surgical clinic for the meeting to be held at Blissfield, May 9. Dr. W. R. Henson, of Shelby, Ohio, has promised to perform a roaring operation and Dr. C. W. Witty, of Elmore, Ohio, will operate on a number of ridgling pigs. The use of the emasculatone will be demonstrated on buck lambs, and Dr. O. W. Schubel, of Quincy, Mich., will dehorn a number of cattle in the recumbent position. Dr. Schubel has worked out a very practical method of restraint for this operation. The clinic will begin promptly at 10 a. m.

Changes at Budapest Veterinary College

The Budapest Veterinary College is now a part of the new Royal Hungarian Palatin Joseph University of Technical and Economic Sciences, according to a decision made by the Hungarian government last summer. The new university is made up of five faculties, each consisting of two divisions. The fourth faculty bears the name, Agricultural and Veterinary Faculty. With this new organization, a few minor changes in the teaching faculty have been made. The Veterinary Division comprises ten departments as follows: Anatomy and Embryology, Prof. Dr. A. Zimmermann; Zoölogy and Parasitology, Prof. Dr. A. Kotlán; Physiology and Histology, Prof. Dr. D. Deseö; Pharmacology and Toxicology, Prof. Dr. J. Magyary Kossa; Pathology, Prof. Dr. Ch. Jármai; Veterinary Medicine, Prof. Dr. J. Marek; Surgery and Ophthalmology, Prof. Dr. E. Guoth; Obstetrics, Prof. Dr. H. Hetzel; Epidemiology and Bacteriology, Prof. Dr. R. Manninger; Animal Husbandry, Prof. Dr. O. Wellmann. Prof. Zimmermann was elected dean of the Agricultural and Veterinary Faculty for the present school year.

A. K.

Old Texas Cattle Brands Pictured

The oldest cattle brands used in Texas are reproduced in a full-page illustration in the March, 1935, *Cattleman*. The brands, dating from 1838 to 1895, were used on cattle in Victoria County, Texas, which has long been outstanding as a producer of live stock. The page is unusually interesting and recaptures for a moment the early romance of the old Southwest.

Memorial Meeting for Dr. F. S. Jones

The memory of Dr. Frederic S. Jones (U. P. '08), who died on October 19, 1934,* was honored at a special meeting held at the Theobald Smith House at the Rockefeller Institute of Medical Research, Princeton, N. J., April 3, 1935. Tributes to the life and works of Dr. Jones were paid by those who had been his colleagues. Dr. Peyton Rous, of the Rockefeller Institute, New York, N. Y., spoke of Dr. Jones' early work, and pointed out those characteristics which gave him his genius for investigation in the field of animal pathology. Dr. Carl TenBroeck, director of the Department of Animal and Plant Pathology, at Princeton, spoke

*Jour. A. V. M. A., lxxxv (1934), n. s. 38 (5), pp. 706-707.

of Dr. Jones' later work with the Institute and the contributions he had made on a number of infectious diseases of animals. Dr. John H. Northrop described the considerable contribution which Dr. Jones made by his investigations of the phenomenon of agglutination.

The meeting was attended by a large number of persons from the various departments of the Rockefeller Institute in New York City and Princeton, from various other educational and research institutions and from a wide circle of professional and personal friends. J. G. H.

Laboratory Technicians Publish Bulletin

The American Society of Clinical Laboratory Technicians now has its own official publication, which has been named the *Bulletin of the American Society of Clinical Laboratory Technicians*. Three numbers of this bimonthly publication have appeared, the third issue being dated March, 1935. On the editorial staff appears the name of Dr. Walter E. King, of Parke, Davis & Co., Detroit, Mich., who is well known to many veterinarians.

Disease Eradication in Puerto Rico

The extension of tuberculosis eradication and Bang's disease work to Puerto Rico is announced by the U. S. Department of Agriculture and is being conducted on a coöperative basis with the Insular Department of Agriculture and Commerce and the Department of Health. Dr. Wilbur McPherson (O. S. U. '15), of the U. S. Bureau of Animal Industry, is in charge of the activities in Puerto Rico, with headquarters in San Juan, the office being located in 309A Ochoa Building. Dr. McPherson was formerly engaged in tuberculosis eradication and tick eradication in Florida.

There are at present five veterinarians engaged in the project. The work now in progress is being done under the Individual Accredited Herd Plan, and is being favorably received by the cattle-owners of the island. The testing for the first two months has revealed an infection of about 2 per cent. There are more than 300,000 cattle in Puerto Rico and, of this number, more than 83,000 are work oxen being used in the sugar industry. About half of the cattle population is of the dairy type.

H. L. V.

Horsemen Fear Equine Encephalomyelitis

A special committee on equine encephalomyelitis, representing the Horse and Mule Association of America, conferred with Dr. J. R. Mohler, Chief of the U. S. Bureau of Animal Industry, in Washington, D. C., January 14, 1935, relative to the advisability of expediting research work on this disease which has threatened the horse industry in certain parts of the United States. The committee consisted of Mr. Thomas P. Cooper, director of the Kentucky Agricultural Experiment Station, Lexington, Ky.; Admiral Cary, T. Grayson, of Washington, D. C., and Mr. Wayne Dinsmore, secretary of the Horse and Mule Association of America. Efforts are being made to enlist the support of the trustees of the University of Illinois to make funds available for conducting further investigations of equine encephalomyelitis at Urbana.

Minnesota's Fight Against Tuberculosis Described

The progress of Minnesota's battle against tuberculosis is recounted in an interesting article by Dr. Charles H. Mayo, of Rochester, Minn., in *Everybody's Health*, the official publication of the Minnesota Public Health Association. Dr. Mayo, who is president of the Association, pays tribute to Dr. J. A. Myers, Professor of Preventive Medicine, University of Minnesota, for his interest in the campaign against bovine tuberculosis.

Long Period of Service for Three Rhode Island Veterinarians

A historical trinity in Rhode Island was dissolved on March 1, 1935, when that state passed a law abolishing all boards and commissions. The trinity consisted of Dr. Charles T. Frey (Ont. '99), Dr. John E. Pollard (Ont. '98) and Dr. Thomas E. Robinson (Ont. '92), chairman, secretary and treasurer, respectively, of the Board of Registration in Veterinary Medicine, all of whom had served in those same positions since the Board was established in 1909.

The first break in the original Board of five members came on Christmas Day, 1928, when Dr. U. S. Richards (Ont. '99) died, and Dr. James M. Armstrong (Harv. '96) took his place. Three years later, Dr. C. Horseman (Amer. '85) died, and Dr. Joseph S. Barber (Chi. '10) was appointed to take his place. The officers themselves had served continuously for 26 years.

Under the new law, the Director of Agriculture will have charge of all examinations for licensing veterinarians. This arrangement is already in effect in several other states.

ORGANIZATION OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION, 1934-1935

(Concluded from page 555, April issue)

RESIDENT STATE SECRETARIES

Alabama .....I. S. McAdory, Auburn.
Arizona .....J. C. McGrath, 1115 E. Van Buren St., Phoenix.
Arkansas .....C. D. Stubbs, War Memorial Bldg., Little Rock.
California .....W. L. Curtis, 1264 W. 2nd St., Los Angeles.
Colorado .....I. E. Newsom, Colorado Agricultural College, Fort Collins.
Connecticut .....Edwin Laitinen, 993 Main St., West Hartford.
Delaware .....R. M. Sarde, Box 5, Camden.
Dist. of Columbia...W. M. Mohler, Bureau of Animal Industry, U. S. Dept. of Agr., Washington.
Florida .....M. W. Emmel, Agr. Exp. Station, Gainesville.
Georgia .....J. Lee Hopping, 1035 Marietta St. N. W., Atlanta.
Idaho .....E. M. Gildow, University of Idaho, Moscow.
Illinois .....C. L. Campbell, c/o Corn Belt Serum Co., East Saint Louis.
Indiana .....Harry W. Brown, 623 W. Main St., Fort Wayne.
Iowa .....C. D. Lee, Veterinary Research Institute, Iowa State College, Ames.
Kansas .....C. B. Clement, Johnson Serum Co., Crane and Jefferson Sts., Topeka.
Kentucky .....E. A. Caslick, Paris.
Louisiana .....Hamlet Moore, 2941 Grand Route Saint John, New Orleans.
Maine .....J. F. Witter, Dept. of Animal Industry, University of Maine, Orono.
Maryland .....M. F. Welsh, College Park.
Massachusetts .....H. W. Jakeman, 44 Bromfield St., Boston.
Michigan .....B. J. Killham, Michigan State College, East Lansing.
Minnesota .....H. C. H. Kernkamp, University Farm, Saint Paul.
Mississippi .....W. L. Gates, Box 417, Clarksdale.
Missouri .....Ashe Lockhart, 800 Woodswether Rd., Kansas City.
Montana .....Hadleigh Marsh, Agr. Exp. Station, Bozeman.
Nebraska .....Frank Breed, 227 N. Ninth St., Lincoln.
Nevada .....L. R. Vawter, University of Nevada, Reno.
New Hampshire ....R. W. Smith, State House, Concord.
New Jersey .....J. G. Hardenbergh, Walker-Gordon Lab. Co., Plainsboro.
New Mexico .....T. I. Means, Box 1174, Santa Fe.
New York .....J. G. Wills, Box 751, Albany.
North Carolina .....J. H. Brown, Tarboro.
North Dakota.....L. M. Roderick, State College Station, Fargo.
Ohio .....W. F. Guard, Ohio State University, Columbus.
Oklahoma .....C. H. Fauks, 1719 S. W. 15th St., Oklahoma City.
Oregon .....W. B. Coon, Forest Grove.
Pennsylvania .....B. Scott Fritz, Dept. of Agr., Harrisburg.
Rhode Island .....T. E. Robinson, 92 High St., Westerly.
South Carolina.....M. R. Blackstock, 157 W. Hampton Ave., Spartanburg.
South Dakota.....G. E. Melody, Gettysburg.
Tennessee .....F. W. Morgan, 1025-27 McCallie Ave., Chattanooga.
Texas .....J. G. Horning, 3611 Willia St., Houston.
Utah .....D. E. Madsen, Utah Exp. Station, Logan.

Vermont .....G. N. Welch, 43 Union St., Northfield.
Virginia .....I. D. Wilson, Virginia Poly. Inst., Blacksburg.
Washington .....C. E. Sawyer, Western Wash. Exp. Sta., Puyallup.
West Virginia .....H. M. Newton, Box 1721, Charleston.
Wisconsin .....J. S. Healy, 330 Federal Bldg., Madison.
Wyoming .....H. D. Port, 304 State Capitol Bldg., Cheyenne.

Resident Territorial Secretaries

Alaska .....J. B. Loftus, Juneau.
Canal Zone.....Lawrence G. Combs, Box 2012, Ancon.
Hawaii .....Bernard A. Gallagher, Box 3319, Honolulu.
Philippine Islands..Victor Buencamino, 1026 Felix Huertas, Manila.
Puerto Rico .....H. L. Van Volkenberg, Agr. Exp. Sta., Mayaguez.
Virgin Islands.....Guy A. Roberts, Christiansted, Saint Croix.

Resident Provincial Secretaries

Alberta .....J. C. Hargrave, Box 673, Medicine Hat.
British Columbia ...J. G. Jervis, Milner.
Manitoba .....J. A. Allen, 1079 Dorchester Ave., Winnipeg.
New Brunswick.....T. Fred Johnston, 117 Leinster St., Saint John.
Nova Scotia.....George Townsend, Box 76, New Glasgow.
Ontario .....R. A. McIntosh, Ontario Veterinary College, Guelph.
Prince Edw. Island..J. R. Cunningham, Summerside.
Quebec .....A. A. Etienne, 1225 Drummond St., Montreal.
Saskatchewan .....Mark Barker, Post Office Bldg., Regina.

Foreign Corresponding Secretaries

Bermuda .....C. J. Cooper, "Kelton," Pembroke.
China .....H. C. Evangelista, Box 1170, Shanghai.
Colombia .....Roberto Plata Guerrero, Apartado 149, Bogota.
Cuba .....Bernardo J. Crespo, Apartado 2518, Havana.
England .....R. W. Tuck, c/o American Consulate, 18 Cavendish Sq., London, W. 1.
Hungary .....A. Kotlán, Royal Hungarian Veterinary College, Budapest, VII.
Jamaica .....Stephen Lockett, Dept. of Agr., Hope, Kingston.
Mexico .....L. Santa Maria, Apartado Postal 2067, Mexico, D. F.
New Zealand .....W. C. Ring, 62 Albert St., Auckland.
Peru .....J. F. Mitchell, c/o Hacienda Pachacayo, Pachacayo.
Saint Kitts.....E. F. Jardine, Box 18, Basseterre.
Scotland .....A. W. Whitehouse, Glasgow Veterinary College, 83 Buccleuch St., Glasgow.

Committee on Policy

H. E. Curry, *Chairman*, 112 Capitol Building, Jefferson City, Mo.

To ancient peoples the horse was sacred. The Scandinavians worshipped it. The Saxon invaders, Hengist and Horsa, bore the horse on their standards and it was symbolized in their names, for the horse was the emblem of warriors.

MISSISSIPPI STATE VETERINARY MEDICAL ASSOCIATION

The twenty-ninth annual meeting of the Mississippi State Veterinary Medical Association was held at Gulfport, January 24, 1935. The meeting was called to order by the President, Dr. A. J. Royal, of Scott.

Three papers were presented at the morning session. Dr. L. A. Mosher, of Atlanta, Ga., discussed "Canine Filariasis," and gave some interesting data on this subject. Dr. C. A. Cary, of the Alabama Polytechnic Institute, spoke on "Veterinary Education," and Dr. F. J. Douglass, of New Orleans, La., gave an instructive talk on "Anaplamosis," which was followed by an animated discussion on the diagnosis and treatment of the disease.

The afternoon session was devoted to Bang's disease. Dr. H. Robbins, U. S. Bureau of Animal Industry, Jackson, talked on "The Technic and Bureau Regulations Governing Bang's Disease," following which the members adjourned to meet eight miles out of Gulfport to observe the bleeding of a herd of cattle according to the technic of the U. S. B. A. I.

The business session accomplished a number of things. A motion to affiliate with the American Veterinary Medical Association was made by Dr. O. N. Norton, of Greenville, seconded by Dr. W. L. Gates, of Clarksdale, and passed by the Association. Members named as delegates to the A. V. M. A. House of Representatives are: Dr. R. H. Stewart, of Jackson, and Dr. C. B. Cain, of State College, alternate. The names of Drs. W. L. Gates, of Clarksdale, C. D. Crawford, of Rolling Fork, and J. T. Alston, of Tupelo, were selected by the Association to be presented to the Governor of Mississippi who will appoint one of this group to the Mississippi Live Stock Sanitary Board for the term beginning January 1, 1936. Four new members also were elected.

Officers for the coming year were chosen unanimously as follows: President, Dr. D. M. Williams, McComb; vice-president, Dr. Andy W. Crawford, Rolling Fork; secretary-treasurer, Dr.

E. H. Durr, Jackson (reëlected). The next meeting will be held at Meridian.

E. H. DURR, *Secretary.*

MARYLAND STATE VETERINARY MEDICAL ASSOCIATION

The winter meeting of the Maryland State Veterinary Medical Association was held in Baltimore, February 7, 1935. The President, Dr. R. C. Reed, of College Park, opened the meeting and delivered his presidential address, in which he stressed the need for greater interest in Association affairs and greater recognition for the profession that would result naturally from such interest.

Of special interest to the members in attendance was the address by Dr. Robert S. MacKellar, president of the American Veterinary Medical Association. Dr. MacKellar emphasized the increasing importance and responsibility of the veterinarian as a holder of the front line of health defense and declared that, with the greater recognition of the profession, will come greater dignity to the calling and remuneration in accordance with the service rendered.

Dr. T. A. Ladson, of Olney, gave a history of drug stimulation of race horses, described methods of detection and outlined the present outlook for the control of this practice. Mr. Louis M. Merryman, of the Agricultural Adjustment Administration, spoke on federal aspects of the Bang's disease-control program and the place of the local practitioner in such a program. Dr. R. S. Amadon, professor of veterinary physiology at the University of Pennsylvania, gave an informative talk on the use and action of various ruminatory drugs, and illustrated his talk with lantern-slides.

Dr. J. P. Turner, of Washington, D. C., gave a brief but interesting report of his experiences as a delegate to the Twelfth International Veterinary Congress, and described the plan of operation and objectives of the A. V. M. A. House of Representatives.

Drs. L. T. Giltner and M. S. Shahan, of the Pathological Division, U. S. Bureau of Animal Industry, Washington, D. C., presented a paper on the recent developments in investigations of equine encephalomyelitis. This instructive paper was supplemented by several reels of motion-picture film, showing horses in various stages of the disease. Since this disease has been prevalent in sections of Maryland for the past two years, an en-

thusiastic discussion ensued. Drs. F. S. Wharton, of Centerville, and W. M. Reynolds, of Chestertown, led the discussion and told of their personal field experiences in handling outbreaks of the disease.

Prof. Joseph Vial, of College Park, and Dr. O. K. Hoffman, of Hagerstown, discussed drouth relief cattle in Maryland. Of 14,000 cattle that were sent into the state from the drouth areas, it is estimated that relatively few animals were lost and that serious outbreaks of sickness were prevented because of the care exercised by official and by local practicing veterinarians.

Among the distinguished guests present were Dr. Taylor P. Rowe, president of the Virginia State Veterinary Medical Association, and Dr. H. C. Givens, state veterinarian of Virginia, who extended an invitation to the members of the Maryland Association to join Virginia and North Carolina in a joint summer meeting at Richmond. The invitation was accepted. As a result, the summer meeting of the Maryland Association, usually held in College Park, will be transferred to Richmond.

Officers elected to serve during the present year are: President, Dr. F. S. Wharton, Centerville; first vice-president, Dr. T. A. Ladson, Olney; second vice-president, Dr. John D. Gadd, Towson; secretary-treasurer, Dr. Mark Welsh, College Park (re-elected). Dr. J. P. Turner, Washington, D. C., was reëlected to the Board of Directors.

MARK WELSH, *Secretary.*

LOUISIANA VETERINARY CONFERENCE

The fourth annual short course for the Louisiana Veterinary Medical Association was held at the Dalrymple Memorial Building, Louisana State University, Baton Rouge, March 6-7, 1935. This short course was made possible by Dean J. G. Lee, Jr., of the School of Agriculture. This year, Dr. E. P. Flower, secretary of the Louisiana Live Stock Sanitary Board, and Mr. Clyde Ingram, of the Poultry Department of the University, contributed to the program. A splendid attendance was recorded.

Dr. James Monroe Smith, president of the University, welcomed the visiting veterinarians, for whom the response was made by Dr. J. A. Goodwin, of New Iberia. President Smith paid high tribute to the late Dr. Harry Morris, who was associated with the University for more than 20 years. This was followed by an address by Dr. H. H. Baur, president of the Association.

Interesting and instructional talks were given by three veterinarians from outside the state: Dr. C. H. Covault, of Iowa State College, Dr. F. D. Patterson, of Alabama Polytechnic Institute, and Dr. T. A. Sigler, of Greencastle, Ind. Dr. Covault's talks were confined to diseases of the dog and dealt with internal parasites and their control, skin diseases, some common digestive disturbances and canine distemper. The members entered freely into discussions of these subjects, particularly the first two. In the afternoon, Dr. Covault gave a demonstration of an oöphorectomy of a bitch.

Dr. Patterson discussed the care and management of poultry and described the more common poultry diseases. He stressed the fact that veterinarians should take advantage of poultry practice and use it as a means of contact with clients. He also presented a paper on "Leukosis," a disease on which he has been doing research work for a number of years.

Dr. Sigler spoke on "Parasites and Their Control in Horses and Mules," "Equine Practice," "Digestive Disturbances of Cattle," and "Reproductive Disorders of Cattle." His discussions were interesting and brought forth many questions. Dr. Sigler also demonstrated the surgical removal of a mummified foetus from a cow.

Dean J. G. Lee, Jr., welcomed the group the second day, and gave an interesting talk on the general agricultural situation, the functions and the results obtained to date by the various governmental agencies. This enlightening talk was well received by the practitioners.

The annual banquet was held at the Heidelberg Hotel on the evening of the first day, with about 40 in attendance. Dr. H. F. Vulliamy, of Donaldsonville, acted as toastmaster. Two wild-game motion-pictures obtained from the Department of Agriculture were shown. Dr. Harry C. Millar, of Asbury Park, N. J., who was in New Orleans for the Mardi Gras, was present at the banquet and made an impromptu talk, as did also Dr. C. T. Dowell, director of the Louisiana Experiment Station, and Mr. B. W. Bateman, Director of Extension. Drs. Sigler, Patterson and Covault were also called upon for short talks.

Officers chosen to serve during the coming year are: President, Dr. H. H. Baur, Monroe; vice-president, Dr. H. A. Burton, Alexandria; secretary-treasurer, Dr. C. M. Heflin, Baton Rouge.

W. T. OGLESBY, *Reporter.*

MAINE VETERINARY MEDICAL ASSOCIATION

The spring meeting of the Maine Veterinary Medical Association was held in conjunction with the Maine Live Stock Breeders' Association, March 28, 1935, during Farm and Home Week at the University of Maine, Orono. Twenty-four members were in attendance. An interesting program was presented. Dr. B. J. Killham, of Michigan State College, spoke on "The Control Problems of Bang's Disease." Dr. M. E. Maddocks, of Augusta, discussed "Common Digestive Troubles of the Horse and Cow," and Dr. H. W. Jakeman, of Boston, Mass., spoke on "The Relationship of the Veterinary Practitioner to the Public." Plans for the summer meeting were discussed.

R. E. LIBBY, *Secretary.*

One Hundred and Fifty Years of Publishing

A publishing firm that can trace its ancestry back in an unbroken line for 150 years is that of Lea & Febiger, Philadelphia, which celebrated on January 25, 1935, a century and a half of continuous activity as publishers. To commemorate the event, this well known firm has published an attractively illustrated and splendidly written booklet, entitled "One Hundred and Fifty Years of Publishing: 1785-1935." The booklet makes absorbing reading, for it records, in one specific line of activity, the history of the United States practically from its very beginning to the present time. In the early days, the firm gave its attention to general literature, publishing the works of Dickens, the Waverley Novels of Scott, the works of Washington Irving, the novels of James Fenimore Cooper and other famous books. Later it began specializing in medical literature and has continued in this field to this day. Since 1859, it has been the publisher in America for Gray's Anatomy, the so-called "Bible of Medicine." It publishes also the *American Journal of Medical Sciences,* now 115 years old. Recently the firm has branched into collateral lines, such as nursing, the biological sciences and veterinary medicine.

"Lassie" Passes at 47

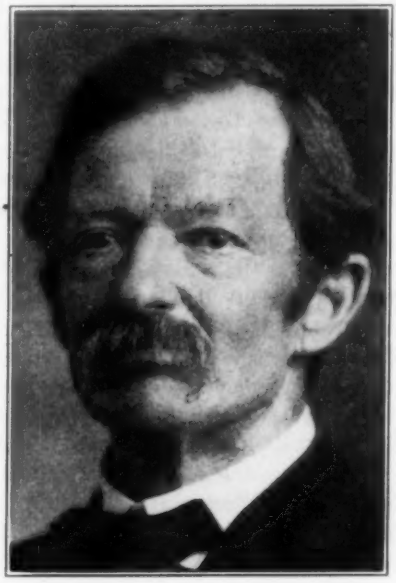
"Lassie," said to be the oldest donkey in Great Britain, was found dead recently. "Lassie," who was 47 years old, was owned by Miss Mabel Bruce, of Bromley. The animal was given to Miss Bruce and her sister in 1888, by a farmer who found the donkey, crippled and helpless, abandoned in a gypsy camp.

Veterinary Record.

NECROLOGY

CHARLES ALLEN CARY

Another member of the Old Guard of the A. V. M. A. has received his summons to join the innumerable caravan—Dr. Charles Allen Cary, outstanding veterinarian of the South for over 40 years. Death came as he slept at his home in Auburn, Ala., some time during the night of April 23, 1935, after a brief illness, reported as influenza, terminated by a heart attack.



DR. C. A. CARY

A familiar figure at A. V. M. A. meetings, which he attended consistently for many years, Dr. Cary usually was a participant in every important debate. He was quite a forceful speaker, and there never was any doubt in the mind of any one as to where Dr. Cary stood on a question. Although it was his lot

to be on the losing side, on more than one occasion, after an important issue had been debated, no one ever said that Dr. Cary was not a good loser.

Dr. Cary was one of the early champions of tick eradication in the South, and his efforts were rewarded when the state of his adoption was freed of the tick in 1930. He consistently championed the inspection of meat and milk by trained veterinarians and was largely instrumental in securing the adoption of inspection ordinances by a number of cities in Alabama. Dr. Cary liked to tell veterinary officials of other states how he managed things in Alabama, which he regarded as a sort of kingdom. The proceedings of the annual meetings of the A. V. M. A., and the U. S. Live Stock Sanitary Association bear testimony on this point. Although his professional activities came first, Dr. Cary was fond of the finer things of life. It was not at all unusual for him to ask to be excused from an evening meeting of some committee or board of which he was a member, in order that he might attend an opera performance.

Born at Millersburg, Iowa, November 27, 1861, Dr. Cary was the son of William and Lucy Ellen Cary. His father was English and traced his line back to the Massachusetts Bay Colony, in 1634. His mother was Scotch-Irish. This rich combination of racial characteristics stood Dr. Cary in good stead through his rather strenuous life. After attending local schools, he entered Iowa State College and was graduated in 1885, with the B. S. degree. Two years later, he completed the veterinary course at the same institution and received his D. V. M. As a student at Ames, he was associated with the late Dr. Sesco Stewart ('85), the late Dr. M. H. Reynolds ('89), Dr. George H. Glover ('86), and Dr. W. B. Niles ('85), all of whom later were to make their marks in the veterinary profession. For about two years, Dr. Cary practiced at Keokuk, Iowa. He then accepted a position as professor of veterinary science at the South Dakota Agricultural and Mechanical College, at Brookings, where he remained until 1892, except for the winter of 1890-91, which was spent in pursuing postgraduate work at the University of Missouri. Early in 1892, he accepted a temporary three-month appointment at the Alabama Polytechnic Institute, at Auburn. The balance of the year was spent in Germany, in further postgraduate work. While there, Dr. Cary met and became intimately acquainted with the late Dr. John W. Adams, who had just been graduated from the University of Pennsylvania and was in Germany, engaged in postgraduate work.

In 1893, Dr. Cary returned to Auburn and joined the faculty of the Alabama Polytechnic Institute, where he remained until his death. When the institution inaugurated a course in veterinary medicine, in 1906, Dr. Cary was made dean of the College of Veterinary Medicine and professor of surgery and milk and meat hygiene.

Dr. Cary joined the A. V. M. A. in 1890, at the first meeting held in Chicago and, at the time of his death, he was one of four members who joined that year who were still on the roll. It is doubtful if any member of the Association held more offices or served on more important committees than Dr. Cary during his 45 years of membership. He first served as resident secretary for South Dakota (1891-92), and upon his removal to Alabama, in 1892, he was appointed resident secretary for that State, a position which he held, except for several interruptions, until 1913. His first committee appointment was as a member of the Committee on Animal Food (1892-93). He was a member of the Committee on Intelligence and Education (1893-94), and member of the Committee on Diseases, for four terms (chairman, 1899-00) in the interval between 1895 and 1904.

Six different A. V. M. A. presidents appointed him a member of the Committee on Resolutions during the period 1897-1928, one term of which (1900-01) he served as chairman. He was a member of the Committee on Publications (1909-10); member of the Special Committee on Advertisements of Veterinary Remedies (1912-13); chairman of the Committee on the Reorganization of the Association (1915-16); member of the Committee on Budget (1919-20); member of the Special Committee on the Revision of the Constitution and By-laws (1922-24); member of the Committee on International Veterinary Congress (1928-34), and member of the Committee on Legislation (1929-34).

During the time that Dr. Cary was serving on these various committees, he frequently served on the Executive Committee, which later was replaced by the Executive Board. He served as a member (1896-97), chairman (1898-99) and again as member (1900-02, 1914-15). He was a member of the Executive Board for District 4, from 1929 until his death. He was elected fifth vice-president of the Association in 1907, and at the meeting in New Orleans in 1919, he was elected to the presidency. He was chairman of the Committee on Credentials of the A. V. M. A. House of Representatives at the time of his death.

Dr. Cary was no less active in the United States Live Stock Sanitary Association. Prior to 1910, this organization was

known as the Inter-State Association of Live Stock Sanitary Boards and Dr. Cary had served (1909-10) as chairman of the Committee on Line and Open Season, which later became the Committee on Tick Eradication. He was actively identified with the Committee on Tick Eradication from 1909 until his death, with the exception of three years. He served as a member of a number of committees: Committee on Legislation (1911-14, 1931-32), Committee on Uniform Health Certificate (1913-15), Committee on Salmon Memorial (1915-16), Committee on Live Stock Diseases (1922-24), Committee on Program (1927-28), Committee on Policy (1930-32) and Committee on Resolutions (1931-32). He was chairman of the Committee on Meat and Milk Hygiene for one term (1931-32). Dr. Cary served three terms as a vice-president (1909-10, 1915-16, 1926-27), and was elected to the presidency at the meeting in 1927.

Dr. Cary served as state veterinarian for Alabama for many years. He was president of the Alabama Live Stock Association for more than a quarter of a century. He was a member of the Alabama Veterinary Medical Association and served, year after year, as secretary-treasurer. He was also secretary-treasurer of the Alabama Veterinary Medical Examining Board. He was a member of the Southern States Veterinary Medical Association and an honorary member of the Iowa Veterinary Medical Association. He was an honorary member of Alpha Psi Fraternity, and his fraternal affiliations included the Masonic Order and the Knights of Pythias. As a churchman, Dr. Cary was a Presbyterian. In addition to these many connections, he found time to serve as a member of the Auburn City School Board for a number of years. He was a member of the Twelfth International Veterinary Congress. Dr. Cary was a prolific writer and was the author of numerous bulletins, pamphlets and scientific articles, dealing with a wide range of subjects in the field of veterinary medicine. He is survived by his widow, two daughters and one son.

WALTER G. MOREHOUSE

Dr. Walter G. Morehouse, of Salem, Ore., died at the Salem General Hospital, March 2, 1935.

Born at Westfield, N. Y., August 17, 1877, Dr. Morehouse was graduated from the New York State Veterinary College at Cornell University, with the class of 1907. In September, 1913, he located at Salem, Ore., and for 17 years was Marion County Veterinarian. He served as president of the Oregon State Vet-

erinary Association for two consecutive terms, and was a member of the Masonic and Elks lodges and of the Salem Rotary Club.

F. W. L.

FRANK REED HANSON

Dr. Frank Reed Hanson, of Sea Cliff, Long Island, N. Y., died suddenly at his home, April 6, 1935.

Following his graduation from the American Veterinary College in 1895, Dr. Hanson became associated in practice with his brother, Dr. Harry D. Hanson (Amer. '89), in New York City. For the past 14 years, he had served as postmaster at Sea Cliff. Surviving Dr. Hanson are his widow (née Ida Ganzenmuller), two sons, two daughters, two brothers and one sister.

H. D. H.

PERSONALS

MARRIAGES

Lt. Edgerton Lynn Watson (K. S. C. '30), of San Francisco, to Miss Zenobia Archibald Baker, at San Francisco, Calif., January 25, 1935.

Dr. H. K. Hudson (K. S. C. '33), of Pontotoc, Miss., to Miss Fern Finney, of Sturgis, Mich., in Memphis, Tenn., March 2, 1935.

PERSONALS

Dr. E. B. McClure (McK. '11) has removed from Davenport, Iowa, to Orion, Ill.

Dr. Matthew J. Eggert (O. S. U. '33) has removed from Irvington, N. J., to Columbus, Ohio.

Dr. M. P. Schlaegel (K. S. C. '20), formerly of Burr Oak, Kan., has removed to Beloit, Kan.

Dr. Harry W. Eggleston (Corn. '14), formerly of Wellsboro, Pa., is now located at Alden, N. Y.

Dr. R. J. Garman (K. C. V. C. '14) has changed locations from Grants Pass, Ore., to Marysville, Calif.

Dr. William Caslick (Corn. '27), has requested a change of address from Schenevus, N. Y., to Otego, N. Y.

Dr. Charles T. Frey (Ont. '99), formerly of West Warwick, R. I., is now living at Hills Grove, same state.

Dr. H. D. Smiley (K. S. C. '30) has requested a change of address from Waynoka, Okla., to Junction City, Kan.

Dr. Francisco R. Taberner (K. S. C. '29) is temporarily receiving his Journal at the State Sanatorium, Norton, Kan.

DR. C. M. PRENTICE (O. S. U. '12) recently opened a hospital for small animals at 1616 Columbus Ave., Sandusky, Ohio.

DR. W. C. SPRINKLE (T. H. '11), formerly of Palmyra, Ill., has opened a new small-animal hospital at Terre Haute, Ind.

DR. PAUL K. BAKER (K. S. C. '17), of Oklahoma City, Okla., is now working on tuberculosis eradication in that state.

DR. J. W. HARKIN (Chi. '14) has been appointed postmaster of Shullsburg, Wis. Dr. Harkin formerly was mayor of Shullsburg.

DR. A. C. TILLMAN (McK. '02) was elected mayor of Earlville, Ill., at the city election held recently. He received 332 votes out of 503 cast.

DR. KARL F. MEYER (Zurich '09), of the University of California, has been nominated for president of the Society of American Bacteriologists.

DR. H. J. METZGER (Corn. '18), formerly of the College of Agriculture of Cornell University, is now located at Lexington, Ky. Address: 1625 Fincastle Road.

DR. J. A. CAMPBELL (Ont. '00), who is curator of the Zoölogical Park, Toronto, Ont., is a director of the American Association of Zoölogical Parks and Aquariums.

DR. C. H. NYE (McK. '15), of Cambridge, Ill., is reported as having purchased a store building on East Street which he will convert into a modern veterinary drug store.

DR. JOHN M. HENDRICKSON (O. S. U. '24), formerly connected with the State School, at Farmingdale, N. Y., has removed to Phoenix, Ariz. Address: 1322 E. Thomas Road.

DR. C. P. HODSON (Iowa '32), of Converse, Ind., has removed to Peru, Ind., where he will continue in general practice. He plans also to establish a hospital for small animals.

DR. CHARLES J. PARSHALL (Corn. '28), of the Division of Veterinary Science, University of California, has taken over the practice of the late Dr. W. H. Pulver, at Brentwood, Calif.

DR. J. S. BENGSTON (McK. '18), of the Chicago Branch Pathological Laboratory, of the U. S. Bureau of Animal Industry, was elected to membership in the Chicago Pathological Society at the April meeting.

DR. JOHN L. MCKITRICK (O. S. U. '33) has resigned his position with the U. S. Bureau of Animal Industry and has resumed general practice at Worthington, Ohio, where his father is an extensive breeder of registered horses and cattle.

DR. R. L. WHITAKER (McK. '17), of Fayetteville, Tenn., was a patient in the Lincoln County Hospital the latter part of March, the result of receiving a broken leg when a cyclone blew down a barn in which he was treating a mule at the time.

DR. W. REID BLAIR (McGill '02), director of the New York Zöological Park, is chairman of the Committee on Express and Transportation of the American Association of Zoölogical Parks and Aquariums in addition to serving the organization as director.

DR. FREDERICK D. PATTERSON (Iowa '23) is the new president of Tuskegee Institute, Tuskegee, Ala., succeeding Dr. R. R. Moton, who has been made president emeritus of the famous Negro school. Dr. Patterson has been head of the agricultural department of the Institute for the past seven years.

DR. R. R. LAUGHLIN (Ont. '13), of South Euclid, Ohio, accompanied by Mrs. Laughlin and their youngest son, enjoyed a three-week vacation cruise in southern waters during April, a short stop being made in the Canal Zone. Dr. N. L. Siplock (Ont. '31), of Ashland, Ohio, looked after Dr. Laughlin's practice during his absence.